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ANNALS of SURGERY

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# THE CAUSES OF DEATH AFTER OPERATION *

## A STUDY BASED ON EIGHT HUNDRED AUTOPSIES

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FROM THE LABORATORIES OF THE MOUNT SINAI HOSPITAL, NEW YORK

About four years ago, at a surgical conference at the Mount Sinai Hospital, there was a discussion on the rôle of pneumonia as a cause of death after operation. One of us was asked to investigate a considerable number of autopsies performed on those who had died after operation and to determine the mortality of post-operative pneumonia. In that analysis pneumonia was a not infrequent cause of death. Frank infection, however, was found far more commonly at autopsy than had been anticipated. An impartial survey of a larger material was decided upon because the subject appeared of sufficient importance to warrant it. Furthermore, post-mortem data from other institutions had to be included in order to obtain more comprehensive statistics of the causes of death after operation.

This report is based on 800 consecutive autopsies performed on patients who died after operation at the Mount Sinai Hospital and at two other representative hospitals in New York. The percentage of autopsies performed on patients who died after operation is high enough to warrant acceptance of the figures as fair indices of the causes of post-operative mortality. The investigation was in no way related to the entirely separate matter of operative mortality, but was solely concerned with the question of why patients die after operation. In every instance, the clinical as well as the autopsy record was studied. Undoubtedly, errors have been made here and there in the interpretation and in the evaluation of various factors. The broad conclusions that can be drawn are therefore of greater importance than the precise figures to be presented.

Autopsies on patients dying within forty-eight hours of operation were excluded. These were mainly cases of operative shock and hæmorrhage, operations as a last resort, or minor procedures on patients suffering from greatly advanced lesions, and we found it impossible to determine the relation of operation to the mortality in most of these very early post-operative deaths. All post-mortem examinations that did not appear to be sufficiently complete to establish a definite cause of death were omitted. As a result autopsies limited to an examination of the head or chest were excluded unless an obvious cause of death was disclosed in the limited examination.

* Read before the New York Surgical Society, January 8, 1930.

Deaths after readmission to the hospital were also omitted unless additional operations were performed. All other cases were listed regardless of how long after operation patients remained alive in the hospitals.

As the analysis progressed we found that the causes of death fell into six categories, and a seventh heterogeneous group:

1. The original disease.
2. Suppuration.
3. Suppuration as a contributing cause, combined with some systemic disease (*e.g.*, generalized arteriosclerosis, diabetes, valvular disease of the heart, chronic nephritis).
4. Suppuration plus pneumonia.
5. Pneumonia.
6. Pneumonia as a contributing cause, combined with some systemic disease as in 3.
7. Miscellaneous, such as embolism, tuberculosis, hæmorrhage, acute yellow atrophy, etc.

Some explanation of the manner in which cases were placed in the different groups is necessary. In a great many instances relatively little consideration was required, for the primary causes of death as disclosed by autopsy were obvious enough. Specifically, whenever there was any doubt as to the rôle of the original disease as compared with infection or a pulmonary complication, the case would be placed in the category of the original disease rather than in any other group. Our purpose was neither to minimize nor to stress the complications of operation but rather to evaluate factors of which we could be reasonably sure.

Death was classified as due to the original disease (Group I) when only the original disease or its extension was disclosed at autopsy. A few examples may be cited: malignancy if the original growth or metastases or complications referable to the tumor were found at post-mortem examination, cases of acute appendicitis or cholecystitis with early peritonitis, pulmonary abscess complicated by empyema after operation, acute osteomyelitis with death from further extension of the disease or from septicemia.

Suppuration (Group II) was termed the cause of death after operation when it was evident and gross at the post-mortem examination and followed operation for a non-suppurating lesion. Purulent peritonitis, gangrene, suppurative septic foci, extravasation of contents of the hollow viscera through suture lines, illustrate the types of lesions classed under infection as the cause of death. If autopsy after an intestinal resection only disclosed a localized plastic peritonitis, the case would not be classified as a death from infection. On the other hand, a frank and undrained collection of pus was termed the cause of death if it were the sole pathological lesion found at autopsy. Cases were placed in Group III when some additional significant lesion was disclosed. Deaths were ascribed to suppuration only when the infection was recent and could be related to the operation or the operative field. Thus a patient who died of pneumonia with complicating

empyema after an interval operation for appendicitis would be placed in the pneumonia group (Group V) if there were no intraperitoneal suppuration.

In the group of suppuration and pneumonia (Group IV) were placed those cases in which both lesions were found at autopsy and in which it was thought that both were important factors in the cause of death. Group V was reserved for pneumonia as the essential cause of death, just as Group II was reserved for infection as the cause of death. A post-pneumonic lung abscess would be placed in the same group as pneumonia. Pneumonia was classed as a contributing cause (Group VI) when systemic disease was found

TABLE I

*Causes of Death—800 Cases*

| | Number of cases | Per cent |
|---|---|---|
| 1. Original disease | 336 | 42.0 |
| 2. Suppuration | 204 | 25.5 |
| 3. Suppuration plus systemic disease | 12 | 1.5 |
| 4. Suppuration plus pneumonia | 32 | 4.0 |
| 5. Pneumonia | 62 | 8.0 |
| 6. Pneumonia plus systemic disease | 28 | 3.5 |
| 7. Miscellaneous | 126[a] | 15.5 |
| | 800 | |

[a] *Miscellaneous causes:*

| | |
|---|---|
| Cardiac disease and arteriosclerosis | 34 |
| Embolism and thrombosis | 25 |
| Ileus (non-peritonitic) | 23 |
| Hæmorrhage | 16 |
| Renal disease | 9 |
| Pulmonary tuberculosis | 8 |
| Duodenal fistula | 4 |
| Acute yellow atrophy of the liver | 2 |
| Pulmonary atelectasis | 1 |
| Ruptured urinary bladder* | 1 |
| Acute appendicitis* | 1 |
| Acute pancreatitis* | 1 |
| Tetanus | 1 |
| | 126 |

* Unassociated with original disease.

at autopsy and the pulmonary lesion was not extensive—for example, a case of generalized arterioscleriosis and chronic nephritis and a terminal bronchopneumonia. On the other hand a terminal bronchopneumonia was not regarded as contributing to the fatal issue in a case of diffuse purulent peritonitis.

Table I was made without reference to the disease for which operation was performed, and without regard to the presence or absence of infection before operation. It is therefore not as significant as the following table, in which the cases are separated into those in which suppuration was and those in which suppuration was not present before operation.

The most striking feature of Table II is the fact that suppuration is the most common cause of death after operations for non-suppurative disease (39 per cent.). The infection was in the immediate neighborhood of the operative field in the great majority of instances. Occasionally the operative

zone was healed or healing and the suppuration, presumably resulting from operation, had localized in some more distant area. Pneumonia was a much more common cause of death (11 per cent.) in the non-infected than in the infected cases (1.5 per cent.). The great bulk of the patients dying after operations for a suppurative lesion died of suppuration (88.5 per cent.). Of considerable importance is the autopsy: evidence that the primary focus of suppuration (appendix, gall-bladder, etc.) had often been adequately cared for at the operations by excision, drainage, or other procedures. The clinical records not infrequently indicate the assumption that the patients had

TABLE II

| Causes of death | 521 Non-suppurative cases | | 279 Suppurative cases | |
|---|---|---|---|---|
| | Number of cases | Per cent | Number of cases | Per cent |
| 1. Original disease | 89 | 17 | 247 | 88.5 |
| 2. Suppuration | 204 | 39 | 0 | |
| 3. Suppuration plus systemic disease | 12 | 3 | 0 | |
| 4. Suppuration plus pneumonia | 22 | 4 | 10 | 3.5 |
| 5. Pneumonia | 57 | 11 | 5 | 1.5 |
| 6. Pneumonia plus systemic disease | 27 | 5 | 1 | .5 |
| 7. Miscellaneous | 110[a] | 21 | 16[b] | 6.0 |
| | 521 | | 279 | |

[a] *Miscellaneous causes:*

| | a | b |
|---|---|---|
| Cardiac disease and arteriosclerosis | 29 | 5 |
| Embolism and thrombosis | 21 | 4 |
| Ileus (non-peritonitic) | 19 | 4 |
| Hæmorrhage | 15 | 1 |
| Renal disease | 8 | 1 |
| Pulmonary tuberculosis | 8 | 0 |
| Duodenal fistula | 3 | 1 |
| Acute yellow atrophy of the liver | 2 | 0 |
| Pulmonary atelectasis | 1 | 0 |
| Ruptured urinary bladder* | 1 | 0 |
| Acute appendicitis* | 1 | 0 |
| Acute pancreatitis* | 1 | 0 |
| Tetanus | 1 | 0 |
| | 110 | 16 |

* Unassociated with origina. disease.

been making satisfactory progress under such circumstances, until the final picture supervened. In other instances an unsatisfactory clinical course was ascribed to some cause far removed from the operative field (pulmonary, cardiorenal) and undrained collections of pus were disclosed at autopsy. The rôle of pneumonia will be discussed in a succeeding paragraph. In this place, we would stress the fact that suppuration was the direct cause of death in the vast majority of patients who died after operations for suppurative lesions, and that the clinical course not infrequently failed to give clear evidence of the true situation.

An analysis of 500 consecutive clinical records was made in the effort to determine how frequently serious post-operative suppuration was assumed to exist in patients operated upon for non-suppurative disease. Of 113 pa-

tients who died primarily of suppuration, forty charts record the diagnosis of infection and fifty have final notes indicating the impression that the cause of death was other than infection. Assuming that the diagnosis of infection was made in the remaining twenty-three cases, and with no desire to place the diagnosis of post-operative suppuration on a statistical basis, the conclusion is inevitable that suppuration after operations for non-suppurative conditions was often not under consideration during life. This should not be interpreted as a criticism of clinical diagnosis. On the contrary, a study of the records warrants the statement that the clinical evidences of suppuration were lacking in very many instances. The conclusion of clinical significance to be drawn is that attention must be focused on suppuration in the operative zone as the probable complication whenever patients are not doing well after opera-

TABLE III

*292 Operations on the Gastro-intestinal Tract*
*(excluding operations for appendicitis)*

| Causes of death | 246 Non-infected cases | | 46 Infected cases | |
|---|---|---|---|---|
| | Number of cases | Per cent | Number of cases | Per cent |
| 1. Original disease | 28 | 11 | 43 | 94 |
| 2. Suppuration | 109 | 45 | 0 | |
| 3. Suppuration plus systemic disease | 9 | 4 | 0 | |
| 4. Suppuration plus pneumonia | 15 | 6 | 2 | 4 |
| 5. Pneumonia | 30 | 12 | 0 | |
| 6. Pneumonia plus systemic disease | 16 | 6 | 1 | 2 |
| 7. Miscellaneous | 39[a] | 16 | 0 | |
| | 246 | | 46 | |

[a] *Miscellaneous causes:*

| | |
|---|---|
| Cardiac disease and arteriosclerosis | 8 |
| Embolism and thrombosis | 8 |
| Ileus (non-peritonitic) | 6 |
| Renal disease | 6 |
| Pulmonary tuberculosis | 5 |
| Hæmorrhage | 3 |
| Duodenal fistula | 2 |
| Acute appendicitis* | 1 |
| | 39 |

* Unassociated with original disease.

tion, regardless of the paucity or absence of signs of suppuration and regardless of an assumed or actual distant complication.

Tables III, IV, V and VI are presented because they are large groups of cases and not because the causes of death were found to be different from those in other groups. The operations performed comprised the very varied list of procedures that would be found in any large series. In the group of exploratory laparotomies, operations for the most part for irremediable lesions, were placed only those procedures consisting in opening the abdomen, exploration, and the removal of specimens in some instances. The relatively unimportant differences in the causes of death between the different groups will be noted. Many individuals well past middle age are included in the

TABLE IV

*76 Operations on the Genito-urinary System*

| Causes of death | 55 Non-suppurative cases | | 21 Suppurative cases | |
|---|---|---|---|---|
| | Number of cases | Per cent | Number of cases | Per cent |
| 1. Original disease | 5 | 9 | 20 | 95 |
| 2. Suppuration | 24 | 44 | 0 | |
| 3. Suppuration plus systemic disease | 0 | | 0 | |
| 4. Suppuration plus pneumonia | 4 | 7 | 0 | |
| 5. Pneumonia | 10 | 18 | 0 | |
| 6. Pneumonia plus systemic disease | 1 | 2 | 0 | |
| 7. Miscellaneous | 11[a] | 20 | 1[b] | 5 |
| | 55 | | 21 | |

[a] *Miscellaneous causes:*

| | |
|---|---|
| Embolism and thrombosis | 4 |
| Cardiac disease and arteriosclerosis | 4 |
| Pulmonary tuberculosis | 2 |
| Renal disease | 1 |
| | 11 |

[b] *Miscellaneous causes:*

| | |
|---|---|
| Ileus (non-peritonitic) | 1 |

TABLE V

*91 Operations on the Liver, Gall Bladder and Ducts*

| Causes of death | 64 Non-suppuration cases | | 27 Suppurative cases | |
|---|---|---|---|---|
| | Number of cases | Per cent | Number of cases | Per cent |
| 1. Original disease | 10 | 16 | 20 | 74 |
| 2. Suppuration | 23 | 36 | 0 | |
| 3. Suppuration plus systemic disease | 0 | | 0 | |
| 4. Suppuration plus pneumonia | 3 | 5 | 2 | 7 |
| 5. Pneumonia | 5 | 8 | 1 | 4 |
| 6. Pneumonia plus systemic disease | 2 | 3 | 0 | |
| 7. Miscellaneous | 21[a] | 32 | 4[b] | 15 |
| | 64 | | 27 | |

[a] *Miscellaneous causes:*

| | |
|---|---|
| Hæmorrhage | 6 |
| Cardiac disease and arteriosclerosis | 5 |
| Ileus (non-peritonitic) | 4 |
| Acute yellow atrophy of the liver | 2 |
| Renal disease | 1 |
| Duodenal fistula | 1 |
| Acute pancreatitis* | 1 |
| Embolism and thrombosis | 1 |
| | 21 |

[b] *Miscellaneous causes:*

| | |
|---|---|
| Cardiac disease and arteriosclerosis | 3 |
| Duodenal fistula | 1 |
| | 4 |

* Unassociated with original disease.

## CAUSES OF DEATH AFTER OPERATION

TABLE VI

*54 Exploratory Laparotomies*

| Causes of death | 47 Non-suppurative cases | | 7 Suppurative cases | |
|---|---|---|---|---|
| | Number of cases | Per cent | Number of cases | Per cent |
| 1. Original disease | 26 | 55 | 6 | 86 |
| 2. Suppuration | 10 | 21 | 0 | |
| 3. Suppuration plus systemic disease | 0 | | 0 | |
| 4. Suppuration plus pneumonia | 0 | | 1 | 14 |
| 5. Pneumonia | 2 | 5 | 0 | |
| 6. Pneumonia plus systemic disease | 3 | 6 | 0 | |
| 7. Miscellaneous | 6[a] | 13 | 0 | |
| | 47 | | 7 | |

[a] *Miscellaneous causes:*

| | |
|---|---|
| Cardiac disease and arteriosclerosis | 2 |
| Hæmorrhage | 3 |
| Embolism and thrombosis | 1 |
| | 6 |

genito-urinary group and the proportionately higher mortality from pneumonia and miscellaneous causes (cardiovascular disease) is probably ascribable to the age factor. Some allowances should be made for a margin of error in the placing of cases in the correct columns in the three groups. For example suppuration in the biliary tract might exist and not be noted at operation for a recently subsided acute cholecystitis, or renal infection may already be present at the time a prostatectomy is performed. It should be stated, however, that, as far as could be learned from the clinical data, cases were placed in their proper categories and that they were placed in the group of suppurative cases whenever there was serious doubt about classification.

Pneumonia is so often in the foreground as a post-operative complication that a special study of its diagnosis, occurrence and significance was made in 500 consecutive cases that came to autopsy. A case was classified as one of pneumonia only when the chart stated "pneumonia" to be the diagnosis. Arbitrarily only those were included in which the diagnosis of pneumonia was made within a week of death, for there might have been difficulties in the post-mortem check-up when the diagnosis had been made at earlier periods. The average time from diagnosis to death was three days. Pneumonia was diagnosed in eighty-four cases and was found at autopsy in fifty-one cases, or 60 per cent. The following significant pathological processes were found in the thirty-three cases in which pneumonia had been erroneously diagnosed:

| | |
|---|---|
| Pulmonary embolism | 2 cases |
| Pulmonary atelectasis | 6 cases |
| Septicemia | 5 cases |
| Hemoperitoneum | 1 cases |
| Empyema secondary to subphrenic abscess | 2 cases |
| Intraperitoneal abscess | 2 cases |
| Diffuse suppurative peritonitis | 15 cases |

No reference will be made to the variety of pathological processes found in the lungs at autopsy to which there were no corresponding references on the clinical charts, for clinical observations may have been made and not recorded, or patients may have been too ill to be subjected to adequate examination of the chest. It is evident, however, that the diagnosis of post-operative pneumonia in sick patients is fraught with difficulty and that classical physical signs of pneumonia do not prove its presence in serious post-operative conditions. The important aspect of the matter lies in the likelihood of committing a grave error by ascribing to pneumonia a serious clinical condition that may be due to suppuration in the field of the operation.

The statement made at the outset, that this investigation was in no way related to a study of operative mortality, will be recalled. We were only concerned with the question of why patients die after operation, not with mortality of various operative procedures. The causes of death in a large series of autopsied cases derived from three representative hospitals have been presented. There is every reason to assume that similar results would be obtained from an analysis carried out at other institutions, for the figures of the three series corresponded closely. Suppuration is the outstanding primary cause of death after operation. The great variety of forms of suppuration noted at autopsy need not be enumerated. There were many instances of single localized collections of pus, accessible to drainage, to which the primary cause of death could be ascribed. What would or might have happened after drainage of such abscesses cannot of course be stated. It is, however, fair to assume that better chances for recovery would have existed.

In our opinion, suppuration following operation for non-suppurative disease is in no way related to errors in technic. On the contrary the highly finished operative technic of today may be the indirect cause. Surgical technic has advanced to such a degree that various formidable operative procedures infrequently practised some years ago are daily occurrences at the present time. Indeed, is it not true that so much reliance is now placed on facile technic that the thought of the patient's margin of safety has to some degree receded into the background? Why will the identical finished technic, applied to an excision of the intestine for example, succeed in one case and be followed by leakage through the suture line and peritonitis in the next? We believe that the answer is to be found in the series of simple exploratory laparotomies, a large proportion of the patients dying of intraperitoneal suppuration. Not only do poorly nourished tissues invite infection; infection is invited in all poorly nourished patients. However, the records also show that suppuration may supervene and be the cause of death after technically satisfactory major operations on patients deemed good surgical risks according to present standards.

# POST-OPERATIVE SHOCK AND SHOCK-LIKE CONDITIONS TREATMENT BY INFUSION IN LARGE VOLUME *

By William F. MacFee, M.D., and Robert R. Baldridge, M.D.
of New York, N. Y.

A number of theories have been advanced to explain shock. One of the most popular of these has been the neurogenic theory which ascribes the fall of blood pressure and consequent shock to exhaustion of the vaso-motor centre. Mitchell, Morehouse, and Keen [1] suggested this possibility near the close of our Civil war. Crile [2] has been its chief sponsor in more recent years.

During the World war, a Research Committee was appointed by the British Government to study traumatic shock. Largely through the activities of this committee, important observations and discoveries bearing upon shock were made. Dale and Laidlaw [3] were able to produce typical shock in animals by the intravenous injection of histamine, and thereby to study shock under experimental conditions. Upon opening animals moribund from this type of shock, they found the heart executing muscular beats of moderate vigor, although the arteries were pulseless. The veins were not distended, and if clamped, they filled very slowly from the periphery. A large part of the blood, in fact, had disappeared from active circulation. It appeared that the weakness of the heart beat was due to a reduced inflow from the veins and not to any essential cardiac deficiency.

In the shock of wounded soldiers Robertson and Bock [4] and Keith [5] have demonstrated a diminution in blood volume, even in the absence of significant hæmorrhage. In experimental shock Gasser, Erlanger and Meek [6] have made similar observations.

The investigations of Cannon, Fraser and Hooper,[7] Taylor,[8] and Robertson and Bock [9] showed that a concentration of corpuscles existed in the capillaries as compared with the veins. Since the ratio of corpuscles to plasma in the veins remained approximately normal, it was assumed that there was a loss of plasma from the capillary channels. These observations have been amply confirmed, both in clinical and in experimental shock.

A large part of the blood which is out of active circulation is to be found, concentrated and stagnant, in the capillaries. Here additional loss of volume occurs by continuous passage of plasma through the capillary walls. Such diminution of volume of actively circulating blood has been called oligæmia, or exæmia, and is now believed to be a very important factor in the production and aggravation of shock.

The inevitable result of oligæmia is impaired effectiveness of circulation and diminution of oxygen supply to the tissues. Because of inadequate

* Read before the Surgical Section of the New York Academy of Medicine, October 4, 1929.

oxygen supply it is probable that changes in osmotic pressure conditions, and other physico-chemical changes not well understood, occur in the capillary areas. That these result in a further loss of circulating fluid is suggested by Hill and McQueen,[10] whose explanation of the sequence of events in shock we find attractive: "With a falling blood pressure as in shock, there is a general constriction of arterioles to maintain sufficient pressure to supply the heart and brain. The general capillary field is sacrificed to the arterial pressure in the brain and heart. A sure Nemesis awaits this method of restoring blood pressure if carried too far. De-oxygenation sets in in the capillary area, the osmotic pressure of the tissue cells rises and these imbibe more fluid, the viscosity of the concentrated blood increases, the capillary wall may suffer and become increasingly permeable. The kinetic pressure available in the capillary area, already a small fraction of a few millimetres of mercury, is inadequate to maintain the blood flow and the heart, so carefully shielded from oxygen want by vaso-constriction elsewhere, finds itself with progressively less and less blood to propel." This chain of events leads to a further decrease in blood pressure and a continuation of the vicious cycle with ultimate complete failure of circulation.

Hill,[11] Krogh,[12] Hooker [13] and Rich [14] have demonstrated that there are numerous capillary channels which under ordinary conditions do not function, but which dilate to receive blood when local tissue needs demand it. The dilation of these capillaries in answer to oxygen want in the tissues probably constitutes an important factor in the loss of blood from the main circulating channels.

Another factor in the production of oligæmia which we believe should be stressed is dehydration. Maintenance of proper blood volume depends to a great extent upon a sufficient reserve of fluid in the tissues or tissue spaces. If a deficiency of circulating blood is threatened it is obvious that the tissue fluids cannot be drawn upon unless they exist in adequate amounts. It is well known that patients who have lost much fluid, as from prolonged vomiting or diarrhœa, are more likely to fall into shock than are patients whose fluid balance has been maintained. Direct loss of circulating fluid through hæmorrhage has long been recognized as an important factor in the production of shock. In the presence of dehydration the loss of a small amount of blood may assume an importance out of all proportion to the amount of blood lost.

A great deal may be done to prevent shock. Diligence in this direction is better than the most skillful treatment after shock is established. An ample supply of fluid should be insured before, after, and sometimes during operation. It is easier to maintain fluid balance than to restore it. Occasionally, however, one is confronted unexpectedly by shock. It may arise under circumstances which are out of control, or it may occur after the most careful preparation and operative procedure.

A plan of treatment for shock has been outlined recently by Cowell.[15] He recommends the following measures: 1, Application of warmth; 2, Pro-

cure mental rest; 3, Relief of pain; 4, Restoration of deficient circulation by giving fluids by mouth, rectally, or by 10 per cent. glucose-saline solution intravenously (one litre in two hours); 5, Increase of intra-cellular oxygenation by insulin hypodermically (five units at beginning and at end of infusion); 6, If operation is needed, by choosing a local or gas-oxygen anæsthesia. Similar suggestions have been made by Fraser[16] in a post-war publication.

In civil practice, cold does not generally play so great a part as in war injuries, but maintenance of warmth is not to be overlooked. Procuring mental rest and relief of pain are important as early measures. In well-established shock, mental unrest and pain usually are strikingly absent. There is instead a remarkable apathy, and sensibility to pain is blunted. Often a needle may be thrust into the vein, or the vein may be cut down upon with little or no disturbance to the patient.

It is chiefly with restoration of deficient circulation that we are concerned. Cardiac stimulants, in the absence of organic cardiac disease, are generally uncalled for. Likewise the use of adrenalin to raise blood pressure is to be condemned. Acting as it does upon the arterioles, its effect is to raise the blood pressure in the arterial tree. But, as pointed out by Cannon,[17] this does not improve volume flow in the capillaries. ". . . Merely a higher arterial pressure is not the desideratum in the treatment of shock, but a higher pressure which provides an increased nutritive flow through the capillaries all over the body. This can be obtained . . . only by increase of the volume flow." Bainbridge and Trevan,[18] injecting adrenalin slowly into the veins of anæsthetized dogs for twenty minutes or longer, at a rate sufficient to raise and maintain blood pressure moderately high, found upon stopping the injection that the animals' blood pressure fell to a low level, with the development of typical shock.

To restore effective circulation it is necessary to restore and maintain the volume of circulating fluid. The importance of an abundant supply of water has been emphasized. Administration by mouth is often impossible or inadequate. The same may be said of rectal instillations. Fluid given subcutaneously, even in large amounts, is likely to be ineffective if shock is at all profound. It is absorbed so slowly that elimination keeps pace with absorption, and the blood volume is not increased. Intravenous injection is the only method by which volume of circulating fluid may be augmented quickly and surely.

For this purpose a number of fluids have been used. Whole blood has many advantages, especially if there has been hæmorrhage. It supplies erythrocytes as well as volume. Furthermore, it does not readily escape from the vessels. Its greatest disadvantages are the lack of ready and adequate supply, and the technical procedures involved in its administration.

The substitutes for blood most frequently employed are solutions of sodium chloride, glucose, acacia, and their combinations. It has long been known that normal salt solution may be safely used for intravenous administration. Clinical experience with it in the treatment of shock has heretofore

been disappointing. Fraser and Cowell,[19] treating shock from war wounds, gave as much as two pints of normal saline solution intravenously to a number of patients. They obtained a temporary rise of blood pressure which was followed by a rapid fall to a level sometimes below the original. Drummond and Taylor [20] record similar conclusions as to the value of salt solutions. Cannon [21] and Bayliss [22] ascribe this failure to the prompt passage of the solution from the capillaries into the tissue spaces. Hypertonic salt solutions proved no better than the normal solution.

Hypertonic glucose solutions have been employed with the idea of increasing blood volume, both directly and by extraction of tissue fluids, and at the same time supplying carbohydrate. It was hoped that glucose might be the means of combating the acidosis of shock. Cannon [23] found, however, that the blood sugar in shock is above normal, and that it is not related to the decreased alkali reserve. Acetone bodies were absent from the urine. The acidosis of shock is not the same as that of diabetes. Thalhimer,[24] Fisher,[25] Beresow,[26] and others, have reported good results by combining the use of insulin with glucose, Padgett and Orr,[27] however, treating experimental shock with glucose solutions, obtained almost identical results whether given with or without insulin. It seems probable that the beneficial effects of glucose are due largely to the water introduced with it.

The principal fault found with the simple crystalloid solutions is that they have generally failed to maintain blood pressure. A primary elevation has been obtained, but has been quickly lost as the solution escaped from circulation.

To overcome this difficulty Bayliss [28] introduced the use of gum arabic. A solution of 0.9 per cent. sodium chloride, containing 6 to 7 per cent. gum arabic has the viscosity of blood and the osmotic pressure of its colloids. It is chemically inert and apparently harmless. It will not pass through the capillary walls and retards the passage of water. Good results from its use have been reported by Drummond and Taylor,[29] who are quoted by Bayliss. Farrar [30] and Randall [31] likewise have found it useful in the field of gynecology and obstetrics. On the other hand, there have been unfavorable reports. Lee [32] mentions two patients in whom he thinks death was hastened by acacia. Hanzlik and Karsner [33] report anaphylactoid phenomena in animals injected with solutions of acacia in saline. Whatever its eventual status in the treatment of shock may be, acacia has not yet come into very extensive use. In addition to the fear of it, whether justified or not, there is the difficulty, in general hospital practice, of securing reliable preparations.

To specifically combat the exæmia of shock we may inject into the vein a fluid, such as blood itself, or the gum-salt solution of Bayliss, with the idea of having it remain in the blood vessels. With the increase of blood volume, blood pressure will be raised, oxygenation of the tissues will be increased, and the vital condition of all the cells in the body will be improved. If a sufficient amount of water can be given afterward, one may expect a lowering of osmotic pressure in the tissues, and the establishment of a balance with

the blood stream. Unfortunately, there are disadvantages which limit the use of these two substances.

Instead of using a fluid which is expected to remain in the blood vessels, we may select a solution such as normal salt or glucose. These, as we know, readily escape from the blood vessels, and are taken up by the fixed tissues, or stored in the tissue spaces. This probably occurs, at least partly, in answer to the physiological demands of the tissues. If solutions of sodium chloride or glucose are to be effective in the treatment of fully developed shock, they should be given in large quantity. In the first place, fluid requirements of the tissues must be met. In the second place, stagnant erythrocytes must be mobilized, viscosity of the blood reduced and blood volume increased. To do this we must fill a circulatory system which, owing to capillary dilation and an increase in number of open capillaries (Krogh[34]), is more capacious than under normal conditions.

We believe that the previous failures with salt solutions have been due to the administration of insufficient quantities. By injecting large amounts of fluid we have had gratifying results in a number of cases treated at St. Luke's Hospital during recent months. In nearly all the cases we have used normal salt solution, often with the addition of varying amounts of glucose. The solution has been given intravenously in amounts ranging from 2000 cubic centimetres to 8000 cubic centimetres at a single injection. The usual amount required has been about 4500 cubic centimetres. It has been given at the rate of about 500 cubic centimetres in ten to twenty minutes. Altogether forty infusions of saline and glucose have been given to thirty patients. Six patients have received a transfusion of blood (500 cubic centimetres) in addition to infusion. As far as we have been able to judge, all the cases treated by infusion were benefited to some extent. In several cases the treatment seemed to be a life-saving measure. In other cases, who eventually died, we have felt that death was due, not primarily to shock, but to the morbid condition which had produced shock.

The post-operative shock-like conditions observed resulted from a considerable variety of diseases. The signs leading to the diagnosis of shock, however, were much the same. The measurable indications of shock following radical mastectomy, for example, differed little from those observed in the "toxæmia" of intestinal obstruction, or of general peritonitis. Delbet[35] and Olivecrona[36] have already called attention to the similarity between traumatic shock and the shock-like condition associated with peritonitis. Similarity in response to treatment has likewise been striking and suggests that a variety of causes may lead essentially to the same condition. Whether it is called "toxæmia" or shock makes little difference so far as the treatment is concerned.

The infusion treatment, in common with other kinds of shock treatment, is most effective when administered early, and when the initiating cause or disease has been removed. Its best results are seen when shock is due simply to a severe operation, uncomplicated by infection, or other factors

not easily eliminated. It is less likely to be permanently beneficial when shock has been produced by a continuing cause, such as peritonitis, or other severe infection. In cases of this type the patient may be brought out of the shock-like state. Then, if his powers of resistance to the disease are good, he probably will go on to recovery. If, however, the infection is overwhelming, shock may again supervene. Infusion can be repeated. The ultimate outcome may be either recovery or death. The infusion treatment in such cases has been useful in so far as it has permitted the patient to combat the disease without the added complication of shock. Infusion, of course, has no specific effect on the underlying disease.

Changes in blood chemistry immediately following infusion have not been striking. Such later changes as have occurred are explainable upon the basis of improved circulation and increased elimination.

The clinical improvement observed in some cases has been impressive. Some of the most interesting changes noted have been the return of normal color to ashen features, often as striking as if a transfusion of blood had been given; a return of tone to the facial muscles, notably those of the eyelids and mouth; a return from lethargy, or even unconsciousness, to relative mental alertness; a slowing of the pulse, with improved quality; the rapid increase of blood pressure, with stabilization near the normal pressure; return of renal function, with disappearance of "urinary suppression"; and, in general, the transition from a precarious condition to one of relative safety.

If given under careful supervision, we believe the administration of the large amounts suggested is safe. In no case have there occurred alarming signs or symptoms of cardiac or respiratory embarrassment. Three patients became a little restless and apprehensive and appeared slightly dyspnœic. In these cases the rate of flow was reduced and symptoms abated. Two patients developed severe chills during infusion. In both of these unbuffered stock solutions of 5 per cent. glucose were being used. The onset of chill occurred in one case after the administration of 1500 cubic centimetres. The infusion was immediately discontinued and no harm resulted. In the second case the chill did not appear until after 3500 cubic centimetres of glucose solution had been given. In this case the glucose was stopped but infusion was continued with normal saline. During the administration of saline solution the chill disappeared. In general, we have had more favorable results when saline was used either alone, or with the addition of prepared ampules of glucose solution. When glucose has been given with the salt solution, we have usually added 50 cubic centimetres of a 50 per cent. solution to 1000 cubic centimetres of normal saline. We have observed little difference whether the saline is given with or without glucose.

We are not unmindful that frequent warnings have been issued stressing the dangers attending intravenous injections of fluids, particularly if given in large amounts. Fatalities following infusion have been recorded in the literature. Acute cardiac dilatation has been cited as a frequent cause of

death. While we have encountered nothing of this kind, we nevertheless would urge constant vigilance. It has been our policy to have the patient under the immediate observation of one of the house staff during the entire infusion. The pulse has been watched at all times, and the blood pressure recorded after the administration of each 500 cubic centimetres of fluid.

Of thirty treated patients, eight died. The causes of deaths were as follows: general peritonitis (three cases), gangrenous ileo-colitis (one case), carcinoma of kidney (one case), intestinal obstruction (one case), liver abscess, tertiary lues, and diabetes mellitus (one case); and carcinoma of breast, bilateral (one case). Death occurred from seven to seventy-two hours after infusion, and from fourteen hours to eight days after operation. Five autopsies were done. In none was there evidence of cardiac dilatation. Two cases, both women, who were moribund when the infusion was begun, showed some pulmonary œdema. The two lungs in one case weighed at autopsy 1200 grams. The lungs of the other case weighed 1310 grams. The average weight for the lungs of a female is given by Morris [37] as 1023 grams.

In the field of animal experimentation there is abundant evidence that any excess of sodium chloride or glucose solution is quickly eliminated from the blood stream. Smith and Mendel,[38] working with rabbits, made intravenous injections of various isotonic solutions in amounts equivalent to the estimated blood volume of the animal. The solutions were injected in two minutes' time. Repeated blood volume determinations were made afterward. In the case of 0.9 per cent. sodium chloride solution, it was found that the larger part of the solution disappeared within the first five minutes after injection, and in the majority of cases, blood volume returned to normal within half an hour.

Lamson and his associates [39] obtained similar results with dogs. Gasser and Erlanger,[40] Smith,[41] and others, have obtained parallel results with glucose solutions.

To establish the relative merits of glucose and sodium chloride solutions in the treatment of shock, further work is needed. Cases of our own who have had blood sugar determinations done before treatment have shown normal, or above normal, content. As a rule plasma chlorides likewise have been normal. It is likely that the greatest value of both glucose and sodium chloride is as an adjunct to the safe administration and retention of water.

The following cases are recorded to illustrate the effects of treatment:

ILLUSTRATIVE CASES

I. Case No. 70388.—E. B., white, female, forty-two years. *Operation.*—February 9, 1929, for congenital anomaly of pancreas, producing obstruction of the common bile duct and chronic pancreatitis.

*Post-Operative Course.*—February 10, 1929, 11 A.M. Face very pale, yellowish color; eyes sunken, half closed; lips and tongue dry; fibrillary twitchings of muscles; completely unconscious; death seems imminent. *Infusion.*—4300 cubic centimetres normal saline solution with 100 grams glucose, and ten units insulin.

| Time | Fluid | Temp. | Pulse | Resp. | Remarks |
|---|---|---|---|---|---|
| 11:30 A.M. | 0 | 106.6 | Too rapid and feeble to count | 5 (irreg., gasping) | Apparently moribund |
| 12 NOON | 1000 | | 180 | 9 (irreg.) | Color better |
| 12:30 P.M. | 2000 | | 168 | 9 (irreg.) | Color good |
| 1:00 | 2800 | 106.3 | 156 | 11 | Voided 100 c.c. (acetone-o) |
| 1:45 | 3200 | 105 | 150 | 13 | Twitchings have ceased; conscious; thirsty |
| 2:45 | 4100 | | 136 | 12 | Vomited |
| 3:15 | 4300 | 104 | 126 | 12 | Comfortable; absolutely conscious |
| Infusion discontinued. | | | | | |
| 9:00 P.M. | | 101.3 | 120 (strong) | 16 | Retaining fluids by mouth |

*Note.*—Blood pressure readings were, unfortunately, not recorded.

*Result.*—Uneventful recovery.

II. Case No. 69571.—White, male, twenty-six years. Adm. B. P. 120/80. *Operation.*—February 27, 1929. Trans-duodenal excision of common duct stone.

*Post-Operative Course.*—February 28, 1929. Transfusion of blood, 600 cubic centimetres. Developed duodenal fistula, second post-operative day, profuse leakage. *Infusion.*—March 8, 1929. Pale, jaundiced gray color; skin, lips and tongue dry; eyes moderately sunken; thirsty; prostrated; mentally depressed and talks about dying. *Infusion.*—4500 cubic centimetres of normal sodium chloride solution.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 1:30 | 0 | 99 | 120 | 20 | 98/86 | See above |
| 1:45 | 500 | | 108 | 20 | 110/80 | |
| 2:00 | 1000 | 98.4 | 108 | 20 | 118/80 | |
| 2:15 | 1500 | | 108 | 14 | 125/80 | Asks for water |
| 2:30 | 2000 | | 106 | 14 | 132/80 | More alert, asks questions about treatment |
| 2:45 | 2500 | | 105 | 12 | 142/85 | |
| 3:00 | 3000 | | 105 | 14 | 140/80 | |
| 3:15 | 3500 | | 106 | 13 | 145/80 | Voided 576 cubic centimetres |
| 4:00 | 4000 | | 106 | 13 | 148/80 | |
| 4:20 | 4500 | | 106 | 13 | 150/82 | |
| Infusion discontinued. | | | | | | |

| Time | Fluid | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|
| 6:00 | | 103 | 16 | 134/80 | |
| 10:00 | | 92 | 18 | 132/78 | |
| March 9, 1929 | | 82 | 16 | 134/80 | Duodenal tube passed beyond fistula for feeding |
| March 10, 1929 | | 90 | 18 | 130/80 | |

| | Urea N | $CO_2$ | Chlor. | Sug. | Bile Ind. |
|---|---|---|---|---|---|
| *Note*—Adm. B. P.—120/80 | | | | | |
| Bl. Chem. 3/8/29 before infusion | 71.5 | 57.6 | 4.5 | | 15 |
| Bl. Chem. 3/9/29 20 hrs. after | 50.0 | 61.7 | 5.25 | 125 | 13 |

*Result.*—Alarming symptoms disappeared after infusion. A second infusion of small amount was given a few days later. Patient made a good recovery.

III. Case No. 72760.—White, female, thirty years. *Operation.*—March 19, 1929. Laparotomy and drainage for general peritonitis, streptococcus hæmolyticus, of undetermined origin.

*Post-Operative Course.*—March 21, 1929. Face generally pale with slight flushing of cheeks; eyes sunken with bluish discoloration about them; lips dry; skin moist; abdomen much distended; constant vomiting of brownish, foul-smelling material; anxious, but mentally clear; critically ill. *Infusion.*—5000 cubic centimetres normal saline.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 9:20 P.M. | 0 | 104.8 | 122 | 23 | 133/80 | Restless, complains of thirst |
| 9:44 | 1000 | | 106 | 26 | 140/74 | Alternately dozing and awake. Asks for water |
| 10:27 | 3000 | | 114 | 36 | 144/74 | |
| 11:15 Infusion discontinued. | 5000 | 105.4 | 116 (Good) | 40 | 148/76 | Again restless, anxious to be left alone, dyspnœic |

| | R. B. C. | Hgb. | W. B. C. | P. | L. |
|---|---|---|---|---|---|
| Bl. Ct. before inf. | 4,600,000 | 90 | 28,800 | 90 | 10 |
| Bl. Ct. after inf. | 3,400,000 | 92 | 12,800 | 88 | 12 |
| Bl. Ct. 10 hrs. after inf. | 4,000,000 | 90 | 10,800 | 88 | 12 |

*Progress.*—March 22, 1929. Condition worse. Gastric lavage at 6 P.M. yielded foul fluid. Passing some gas by rectum. Two watery stools. Distention still very great.

*Second Infusion.*—March 22, 1929. 3500 cubic centimetres 5 per cent. glucose (unbuffered), plus 600 cubic centimetres normal saline.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 7:30 P.M. | 0 | 103.0 | 96 (weak) | 24 | 110/64 | Voided 700 cubic centimetres during infusion. Had chill toward end |
| 9:00 | 4100 | 104.8 | 136 (good) | 40 | 165/80 | |

Infusion ended.

| | R. B. C. | Hgb. | W. B. C. | P. | L. | Bl. U. N. | $CO_2$ | Sug. | Chlor. |
|---|---|---|---|---|---|---|---|---|---|
| Before inf. | 4,800,000 | 92 | 11,000 | 90 | 10 | 35.7 | 37.2 | 167 | 5.85 |
| Immed. after | 4,200,000 | 90 | 6,000 | 84 | 16 | | | | |
| 12 hrs. after | | | | | | 29.4 | 39 | 125 | 6. |

*Result.*—Gradual improvement after second infusion to complete recovery.

IV. Case No. 71898.—White, female, forty years. Adm. B. P. 126/82. *Operation.*—April 2, 1929. Supra-vaginal hysterectomy for fibromyoma.

*Post-Operative Course.*—April 3, 1929. Developed symptoms and signs of peritonitis. *Infusion.*—April 4, 1929. Definite severe general peritonitis, streptococcus hæmolyticus. Infusion of 5000 cubic centimetres normal saline solution.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 2:55 P.M. | 0 | 104.2 | 126 | 30 | 98/72 | Face pale and moist. Tongue and lips parched. Distended; apathetic |
| 3:30 | 2500 | | 120 | 27 | 120/65 | |
| 4:05 | 5000 | 102.4 | 120 | 32 | 135/70 | Face flushed; pulse well sustained. Voided 250 cubic centimetres |
| Infusion discontinued. | | | | | | |
| 8:00 | | 102 | 103 | 26 | 128/80 | |

| | R. B. C. | Hgb. | W. B. C. | P. | L. | B. U. N. | $CO_2$ | Sug. | Chlor. |
|---|---|---|---|---|---|---|---|---|---|
| Immed. before inf. | 5,800,000 | 102 | 28,600 | 92 | 8 | 18.5 | 40.9 | 133 | 6.25 |
| Immed. after inf.. | 4,000,000 | 84 | 28,800 | 90 | 10 | 12.5 | 39 | 118 | 6.5 |

*Second Infusion.*—April 7, 1929. 2000 cubic centimetres saline plus 1000 cubic centimetres 5 per cent. glucose.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 5:55 P.M. | 0 | 104.4 | 160 | 44 | 94/66 | Face pale. Breathing shallow. Mouth open |
| 6:40 | 3000 | 103.4 | 146 | 51 | 116/62 | Face flushed |
| Infusion discontinued. | | | | | | |
| April 8, 1929 | | 103 | 142 | 44 | 106/70 | Some gas expelled |

*Third Infusion.*—April 9, 1929. 4500 cubic centimetres saline with 50 grams glucose and 5 cubic centimetres digitan.

| | | | | | | |
|---|---|---|---|---|---|---|
| 9:00 A.M. | 0 | 106.4 | 150 | 40 | 70/? | Semi-conscious, condition grave |
| 12:00 NOON | 4500 | 102 | 130 | 45 | 114/66 | Fully conscious, much improved |
| Infusion discontinued. | | | | | | |
| 8:00 P.M. | | 101.6 | 112 | | 112/70 | |

*Fourth Infusion.*—April 10, 1929. 5,500 cubic centimetres saline with 50 grams glucose.

| | | | | | | |
|---|---|---|---|---|---|---|
| 9:50 A.M. | 0 | 104.8 | 135 | 18 | 64?/50? | In coma, face white. Eyes fixed; apparently dying |
| 12:15 P.M. | 5500 | | 134 | 20 | 80/50 | |
| Infusion discontinued. | | | | | | |
| 6:00 | | | | | | Unconscious. Shows some œdema of legs |

*Result.*—April 11, 1929. Died.

*Autopsy.*—80 cubic centimetres fluid in right pleural cavity; 60 cubic centimetres fluid in left. Both lungs show moderate œdema and advanced congestion in both inferior lobes. Right lung weighs 710 grams. Left lung weighs 600 grams. *Heart* is normal. *Abdomen.*—Acute fibrinous peritonitis.

V. Case No. 71944.—White, male, thirty-six years. *Operation.*—April 6, 1929. Gastro-enterostomy for duodenal ulcer. Cholecystectomy for chronic cholecystitis.

*Post-Operative Course.*—April 7, 1929. Twenty-four hours after operation, patient appeared quite ill; frequent vomiting; profuse sweating; face pale, lead gray color; eyes sunken; extremely prostrated; very apathetic; response to questions is slow but accurate. *Infusion.*—April 7, 1929. 5000 cubic centimetres normal saline solution.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 12:40 P.M. | 0 | 100.3 | 120 (thin) | 18 | 110/85 | Drowsy |
| 12:55 | 1000 | | 102 | 22 | 125/75 | Color better. More alert |
| 1:10 | 2000 | | 99 | 21 | 130/75 | |
| 1:25 | 3000 | | 98 | 24 | 135/70 | |
| 1:40 | 4000 | | 100 | 24 | 140/75 | Talkative |
| 2:05 | 5000 | 100.3 | 96 | 21 | 140/75 | Marked clinical improvement |
| Infusion discontinued. | | | | | | |
| April 8, 1929 | 3 P.M. | | | | 128/75 | |
| April 9, 1929 | 3 P.M. | | | | 128/75 | |

*Result.*—Recovery.

VI. Case No. 75570.—White, female, thirty-four years. *History.*—Seven days after delivery of a full-term baby, patient developed symptoms of acute intestinal obstruction. *Operation.*—September 27, 1929. (Three days after onset of symptoms.) Laparotomy revealed obstruction of upper small intestine due to volvulus. Adhesions freed and obstruction relieved with minimum of trauma and very little loss of blood.

*Post-Operative Course.*—Two hours after operation patient presented the typical picture of shock. *Infusion.*—2000 cubic centimetres normal salt solution (two hours after operation).

| Time | Fluid | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|
| 3:30 P.M. | 0 | 140 Weak | 24 | 80/64 | Unconscious; condition poor |
| 3:40 | 500 | 124 Slight Imp. | 24 | 92/64 | Cold |
| 3:50 | 1000 | 120 Fair | 24 | 110/68 | Warmer; conscious |
| 4:00 | 1500 | 112 Good | 24 | 110/64 | Conversing |
| 4:10 | 2000 | 110 Strong | 24 | 108/66 | Asks for water |
| Infusion discontinued. | | | | | |
| September 29, 1929 | | 84 | | 125/80 | |

*Note.*—Urinary output during night before operation was 45 cubic centimetres. Catheterization two hours after infusion yielded 290 cubic centimetres.

*Result.*—Patient made a good recovery.

VII. Case No. 74384.—White, male, forty-seven years. *History.*—Patient entered hospital with uniformly enlarged thyroid; general symptoms of hyperthyroidism; heart enlarged; auricular fibrillation; basal metabolism, plus 96. After one month of medical treatment, basal metabolism dropped to plus 15, and patient was operated upon. *Operation.*—September 5, 1929. Subtotal thyroidectomy.

*Post-Operative Course.*—Twelve hours after operation patient was extremely prostrated; pale; lips and tongue dry; very dyspnœic, breathing with mouth open; thirsty but too dyspnœic to drink, except small sips. Pulse rapid and weak, but regular.

*Infusion.*—3600 cubic centimetres normal salt solution twelve hours after operation.

| Time | Fluid | Temp. | Pulse | Resp. | B. P. | Remarks |
|---|---|---|---|---|---|---|
| 9:00 P.M. | 0 | | 156 | 40 | 90/50 | Condition critical |
| 10:30 | 3600 | | 144 | 32 | 155/65 | Face flushed; respiration easy |

*Note.*—Most striking features were return of excellent color to face, improvement in quality of pulse, and relief of dyspnœa. At end of infusion patient asked for water and drank a full glass without difficulty. Twenty-four hours after infusion the blood pressure was 150/65.

*Result.*—Recovery.

## SUMMARY

1. Whatever the absolute cause of shock may be, the essential fact of shock is de-oxygenation of the body tissues.

2. De-oxygenation occurs from impairment of circulation.

3. The impairment of circulation results from diminution of blood volume *in circulation*. This loss is due to stagnation of blood in the capillary areas, and to escape of plasma from the capillary channels. Hæmorrhage and dehydration are frequent factors.

4. To rationally combat shock, restoration of volume of blood in effective circulation is of first importance.

5. Physiological sodium chloride solution has heretofore been tried as a medium to replace lost volume and has been generally discarded.

6. By using physiological sodium chloride solution, with or without glucose, in amounts much larger than have usually been employed, we have consistently obtained gratifying results in the treatment of shock.

7. The danger of producing acute cardiac dilatation or pulmonary œdema has not been apparent in a relatively short series of cases.

8. We practise and urge, however, constant vigilance during the administration of large amounts of solution.

9. We believe that the results obtained to date with this method of treatment warrant continuation of its use.

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# BRONCHIAL INJURY AND REPAIR *

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The reparative processes of bronchi following an injury have been infrequently considered either clinically or experimentally. However, this problem has been encountered quite often with the advent of lung surgery. In performing lobectomies and pneumectomies much difficulty has been experienced in obtaining a satisfactory closure of the bronchial stump. Bettman [1, 2] (1924) found that in a successful closure of the stump, the healing process consisted of peribronchial tissue, without which it was practically impossible to close a primary bronchus permanently. Also that a pneumectomy could not be performed successfully because of the lack of enough tissue to cover the stump.

Robinson and Sauerbruch [3] (1909), quoted by Bettman, make the following statements: "Experimental removal of a single lobe of the lung in dogs is almost always successful. The reverse is true following the complete extirpation of the lung, a few animals dying from infection, but more dying on the sixth to eighth day due to opening of the bronchus with resultant pressure pneumothorax and mediastinal emphysema." Willy Meyer's [4] (1909) successful closure of the stump of one or more lobes did not show healing of the bronchus to be playing a part in the closure. It is to be noted that a generous stump was left in Lilienthal's technic for lobectomy, as was also the case in Joannides' [6] experiments.

Again, the slowness to heal of many of the larger bronchial fistulæ is perhaps somewhat related to the problem of bronchial repair. The experiences of Halstead and Thurston,[7] Eggers,[8] Graham,[9, 10] Keller, [11] and others lead one to believe this to be true. Too often, operative interference has only caused a prolongation of the existence of the fistula. The difficulty and danger in closing some fistulæ is so great that the advice, "Hands off," for a period of months or even years, has been given [12] with the hope that a spontaneous closure may result.

With these problems in mind the task was entered upon to study experimentally, how the air passages, mainly bronchi, reacted to a severe injury, and the stages of healing in the repair of the damage.

Experimental.—Dogs were used exclusively; the weights ranging from 12 to 15 kilograms. A preoperative dose of morphine grains ½ per kilogram and atropine grains 1/150 per kilogram was given one-half to one hour before operation. No other

* This work has been conducted under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

anæsthesia was employed. The site of election for cauterization was usually the medial secondary bronchus of one of the lower lobes, this location being easily accessible and the optimum for observation. Occasionally the bronchus of the accessory lobe (subcardiac) was used.

Two types of cauterizing agents were employed, *viz.*: actual thermal cautery, and silver nitrate. For thermal cautery, four or five coils of a small wire attached to a nine-volt battery with a rheostat in the circuit were used. The coils were hot but not red, so that the effect was coagulating and not searing. For the silver nitrate cautery the stick form of the chemical was used, the stick being secured at one end of a brass tube by means of adhesive tape (Fig. 1).

*Procedure.*—About one-half to one hour after the morphine had been given, the dog was found to be in a stuporous state. (If too much morphine is given the dog will be hyperirritable.) With the animal secured in a dorsal position and a mouth gag in place, a bronchoscope (fashioned after Jackson's type) was introduced and carried down to one of the primary bronchi, and the medial secondary bronchus of one of the lower lobes located. The thermal cautery was then introduced under direct vision into the bronchus for a distance of one to two centimetres (it fit fairly snugly) and allowed to remain for ten seconds. After removal of the cautery, a white eschar 75 centimetres in width, was seen encircling the bronchial lumen.

In the case of silver nitrate, the stick was introduced into the medial secondary bronchus of the lower lobe (in place of the thermal cautery) and allowed to remain for one minute. On removing the cautery a greenish-white area of corrosion of about 1 to 1.5 centimetres in width was found encircling the bronchial lumen.

In the first few experiments, the silver nitrate was applied to both lower lobes. The dogs were unable to endure this amount and usually died on the second or third day of an intense bilateral pneumonia. In most of the work silver nitrate was used on one side and thermal cautery on the other.

The dogs were caged immediately following the procedure and sacrificed by electrocution[12] at intervals of: thirty minutes, three hours, one day, one week, two weeks, three to four weeks, six weeks and eight weeks, at which time gross and microscopic studies were carried out. For microscopic study hæmatoxylin and eosin and elastic tissue stains were made. In two dogs the bronchus of the accessory lobe was selected as the site of cauterization. Thermal cautery was repeatedly applied for ten-second periods at intervals of one to three weeks. Changes were noted from time to time by means of bronchoscopic examination (see protocols).

In obtaining the following results, a high rate of mortality was at times encountered. This is evidenced by the fact that, out of seventy dogs receiving cauterization, thirty-three died and thirty-seven were sacrificed. Part of this high mortality was not due to cauterization alone but to a combination of cauterization and pre-existing pneumonia. Also, as stated above, the first six dogs receiving silver nitrate on both sides, died within twenty-four to forty-eight hours. The mortality rate was low where either type of cautery was used alone and applied at only one location.

Microscopic studies were made on fifty-two of the seventy dogs. Thus several specimens of each stage were obtained, thereby eliminating the danger of drawing biased conclusions.

Typical protocols are as follows:

Protocol I.—*Thermal Cautery.*—August 24, 1928. Dog No. 895, weighing 12 kilograms, was given 3.0 grains of morphine and 0.08 grams of atropine one-half hour before operation. With the animal secured in a dorsal position and a mouth gag in place, the bronchoscope was introduced into the larynx and carried down into the right primary bronchus. The medial secondary bronchus of the right lower lobe was visualized. The thermal cautery was directed into this bronchus for a distance of 1 to 2 centimetres and allowed to remain for ten seconds. Following removal of the cautery, a white eschar was noted encircling the lumen of the air passage. The bronchoscope and

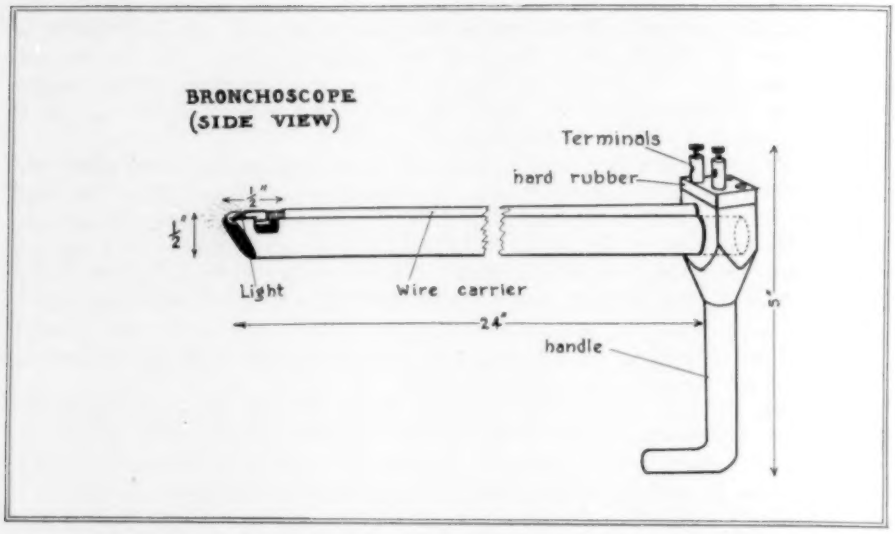


FIG. 1A.—Diagrammatical illustration of apparatus used in producing an injury to the bronchus.

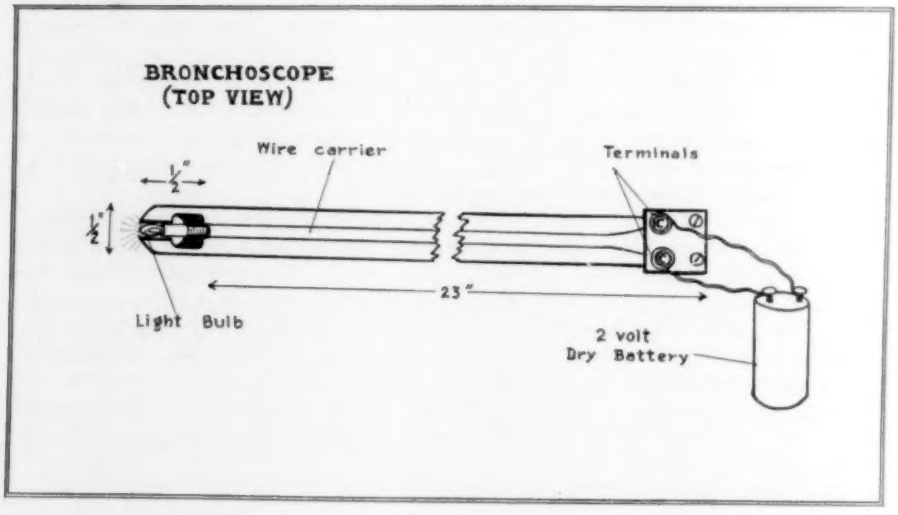


FIG. 1B.

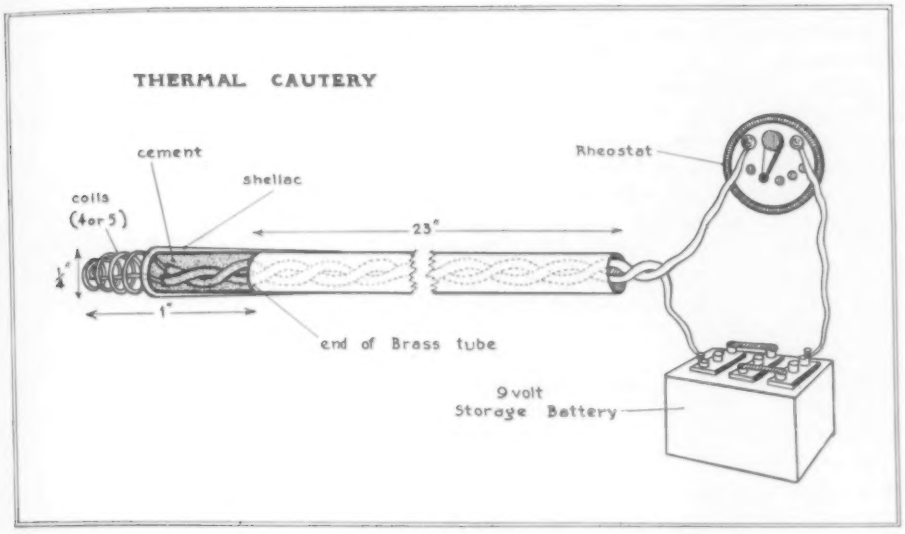
THERMAL CAUTERY
cement
shellac
coils
(4or5)
23"
Rheostat
1"
end of Brass tube
9 volt
Storage Battery

FIG. 1C.

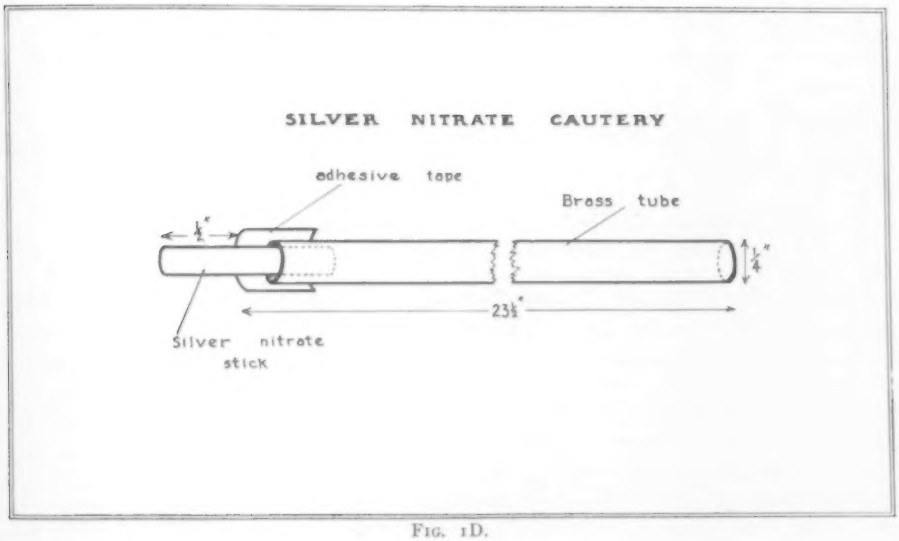
SILVER NITRATE CAUTERY
adhesive tape
Brass tube
23½"
Silver nitrate
stick

FIG. 1D.

mouth gag were removed, and the dog returned to his cage in a more or less stuporous state.

August 25, 1928, dog very quiet and listless. Did not eat. No coughing.

August 26, 1928, dog remained quiet and ate little. No coughing.

August 27, 1928, better today. Ate more although he continued to be quiet.

August 28, 1928, more active and ate well. No coughing.

August 29, 1928, appeared improved. Took food well. Occasional non-productive cough noted.

August 31, 1928, condition unchanged.

September 1, 1928, appeared practically normal although occasional non-productive cough was still present.

September 2, 1928, to November 24, 1928, animal appeared in good health.

November 24, 1928, sacrificed by electrocution.

PROTOCOL II.—*Silver Nitrate Cautery.*—August 22, 1928. Dog No. 896, weighing 13.5 kilograms, was given 3.5 grains of morphine and 0.09 grains of atropine one-half hour before operation. The animal was secured lying on its back and a mouth gag inserted. The bronchoscope was introduced into the trachea and carried down to the right primary bronchus. The medial secondary bronchus of the right lower lobe was visualized and the silver nitrate stick directed into it for a distance of one inch. It was allowed to remain for one minute, during which time it was slowly rotated for a two-fold purpose, *viz.*: to cauterize the entire circumference of the wall, and to keep the silver nitrate stick from adhering to the bronchial wall and thus losing it on attempting withdrawal. After removal of the cautery the bronchial wall was found to be much corroded. The bronchoscope and mouth gag were removed and the dog returned to its cage, still in a stuporous state.

August 23, 1928, dog very quiet. Did not eat. No coughing or barking. Appeared quite ill.

August 24, 1928, little change noted.

August 25, 1928, occasional non-productive cough observed. Ate very little.

August 27, 1928, some improvement noted. Ate more and was more active. Cough still present.

August 28, 1928, took food quite well. Still coughing.

August 30, 1928, improved. Less cough noted.

September 1, 1928, quite well. Took food very well and only very occasional cough noted.

September 2, 1928, to November 22, 1928, animal appeared in good health.

November 22, 1928, sacrificed by electrocution.

PROTOCOL III.—*Repeated Thermal Cauterization.*—August 27, 1928. Dog No. 935, weighing 12 kilograms, was given three grains of morphine and 0.08 grains of atropine one-half hour before operation. The animal was secured in a dorsal position and a mouth gag inserted. The bronchoscope was introduced into the trachea and carried down into the right primary bronchus. The bronchial opening to the accessory lobe was visualized. The thermal cautery was introduced into it for a distance of 0.5 inches and allowed to remain for ten seconds. Following removal of the cautery, the entire circumference of the bronchial wall presented a white eschar about .75 centimetre in width. The bronchoscope and mouth gag were removed and the dog returned to its cage. Its behavior for the week following was no different than that of dog No. 895, described above.

September 4, 1928, X-rays showed no demonstrable pathological changes in the lungs. The dog was given 2.5 grains of morphine and bronchoscoped one-half hour later. The accessory lobe bronchus, at the site of injury, was found much reddened, swollen, œdematous and bleeding. Thermal cautery was applied for five seconds to the

site of original cauterization. The reaction of the dog during the following week was very similar to that following the first cauterization.

September 11, 1928, X-rays again showed no demonstrable pathological changes. Bronchoscopy (following morphine grains 2.5): The origin of the accessory lobe bronchus was found much swollen and œdematous with the size of its lumen diminished. No cautery was applied at this time.

September 12, to 19, 1928, dog appeared in good health; eating well and no coughing.

September 19, 1928. Bronchoscopy (following morphine grains 2.5): Much swelling and œdema of site cauterized. No cautery applied. Dog appeared normal during the week following.

September 27, 1928. Bronchoscopy (following morphine grains 2.5): The origin of the accessory lobe bronchus was found partially stenosed. It did not appear acutely inflamed. Thermal cautery was applied for ten seconds.

September 27, to October 4, 1928, dog was quiet for the first day following cauterization, then appeared normal again, eating well and not coughing.

October 4, 1928. Bronchoscopy (following morphine grains 2.5): The origin of the accessory lobe bronchus was filled with a muco-purulent discharge. Some reddening of the mucosa was present. No cautery applied. During the two months following the dog appeared normal and healthy.

December 6, 1928. Bronchoscopy (following morphine grains 2.5): The origin of the bronchus cauterized showed definite marked stenosis. Thermal cautery was applied for ten seconds. The dog was quiet for a day, then appeared quite normal for the three weeks following.

December 31, 1928. Bronchoscopy (following morphine grains 2.5): An increase in the degree of stenosis of the bronchus was noted. No inflammation was seen. Thermal cautery applied for ten seconds. The dog was quiet for a day, then appeared normal for the three weeks following.

January 22, 1929. Bronchoscopy (following morphine grains 2.5): The origin of the accessory lobe bronchus was found markedly stenosed. The lumen was about 2 millimetres in diameter. Thermal cautery was applied for ten seconds. The dog was quiet for a day, then appeared normal during the two weeks following.

February 6, 1929. Bronchoscopy (following morphine grains 2.5): The origin of the accessory lobe bronchus was completely stenosed. No cautery applied. Dog appeared normal for two weeks following.

February 20, 1929. Bronchoscopy (following morphine grains 2.5): The bronchial origin was completely stenosed as seen before. No cautery applied.

June 5, 1928, sacrificed by electrocution.

RESULTS.—*Thermal Cautery*—Thirty-Minute Specimen. *Gross Pathology.*—The surface of the lung lobe appeared normal. The bronchus presented a white eschar at the site of cauterization. Surrounding this was a zone of hyperæmia and œdema extending for a distance of 0.5 centimetre away from the bronchial wall.

*Microscopic Pathology.*—The bronchial wall was entirely necrosed, except for a small part of the cartilage. The surrounding parenchyma presented dilated vessels. Much œdema with but little hæmorrhage was present.

*Silver Nitrate Cautery*—Thirty-Minute Specimen. *Gross Pathology.*—A slight injection was noted on the surface of the lung lobe opposite the site of cauterization. The bronchial wall showed much corrosion at the site of cauterization around which much hyperæmia and œdema were seen extending for 2 centimetres away from the bronchial wall.

*Microscopic Pathology.*—The bronchial wall was entirely necrosed except for a small part of the cartilage. There was also necrosis of some of the parenchyma surrounding the bronchus. Distal to this the vessels were dilated with much œdema and some hæmorrhage into the alveoli.

*Thermal Cautery*—Three-Hour Specimen. *Gross Pathology.*—A circular area of injection two to three centimetres in diameter was noted on the surface of the lobe opposite the site of cauterization. The bronchus presented a white eschar at the site of injury, with hyperæmic changes surrounding it for a distance of 1 centimetre.

*Microscopic Pathology.*—The bronchial wall had changed but little from the thirty-minute stage. Surrounding this, a pneumonic process was seen becoming manifested by the beginning infiltration of polymorphonuclear leucocytes with also a few lymphocytes. Much hæmorrhage was present, with much blood pigment scattered through the parenchyma.

*Silver Nitrate Cautery*—Three-Hour Specimen. *Gross Pathology.*—A circular area of injection 3 centimetres in diameter was noted on the surface of the lobe opposite the

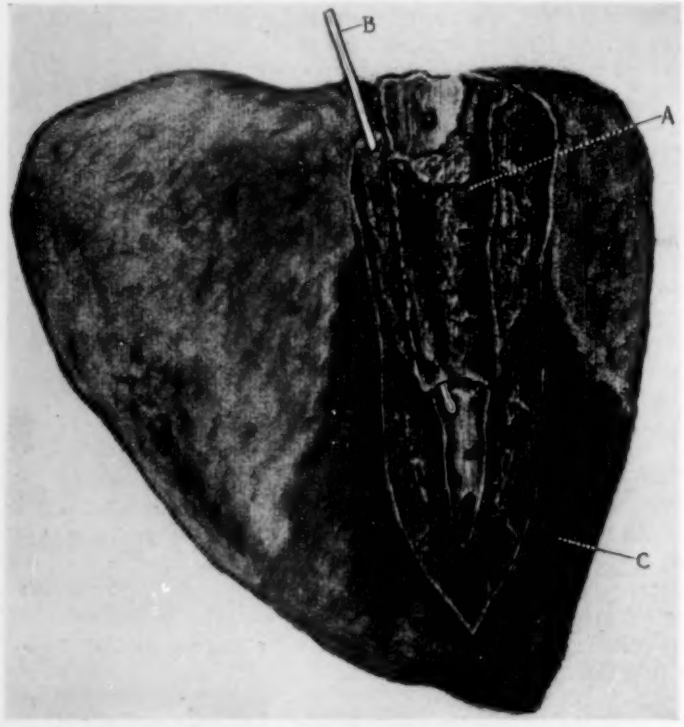


FIG. 2.—Dog No. 200A. Right lower lobe of dog dying from hæmorrhage following cauterization with silver nitrate, one day duration. Note—(A) Corrosion of bronchus at site of cauterization. (B) Probe in pulmonary artery which ruptured into bronchus. (C) Large hæmorrhagic infarct distal to site of cautery.

site of cauterization. Much corrosion of the bronchial wall at the site of injury was seen, with surrounding hyperæmic changes for a distance of 2 centimetres.

*Microscopic Pathology.*—The bronchial wall appeared the same as in the thirty-minute stage. Surrounding this the same changes as seen in the thermal cauterization of the three-hour stage, were noted. However the changes were more marked here and extended farther away from the necrosed bronchial wall.

*Thermal Cautery*—One-Day Specimen. *Gross Pathology.*—The lobe surface was injected slightly more than in the three-hour stage. The bronchial wall was seen beginning to slough at the site of cauterization, with some associated ulceration and hæmorrhage.

*Microscopic Pathology.*—The necrosed bronchial wall was seen beginning to sequestrate. Surrounding the bronchial wall both leucocytes and lymphocytes had

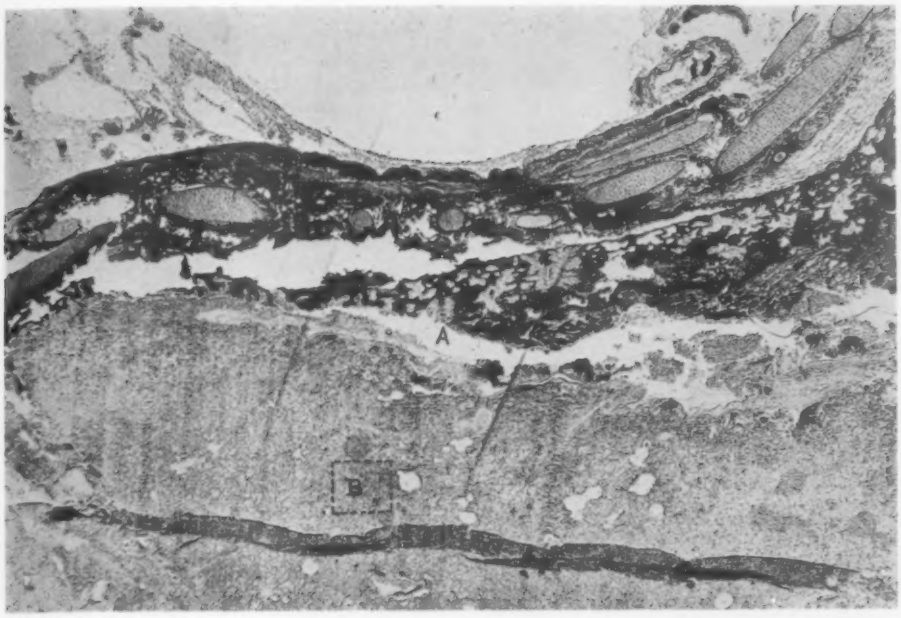


FIG. 3.—Dog No. 870. Microscopic appearance of bronchial wall and surrounding parenchyma following silver nitrate cauterization, one-day duration. Note (A) Separation of necrosed bronchial wall and adjacent parenchyma from surrounding lung tissue. (B) Location for more detail study. See Fig. 4. (x 35.)

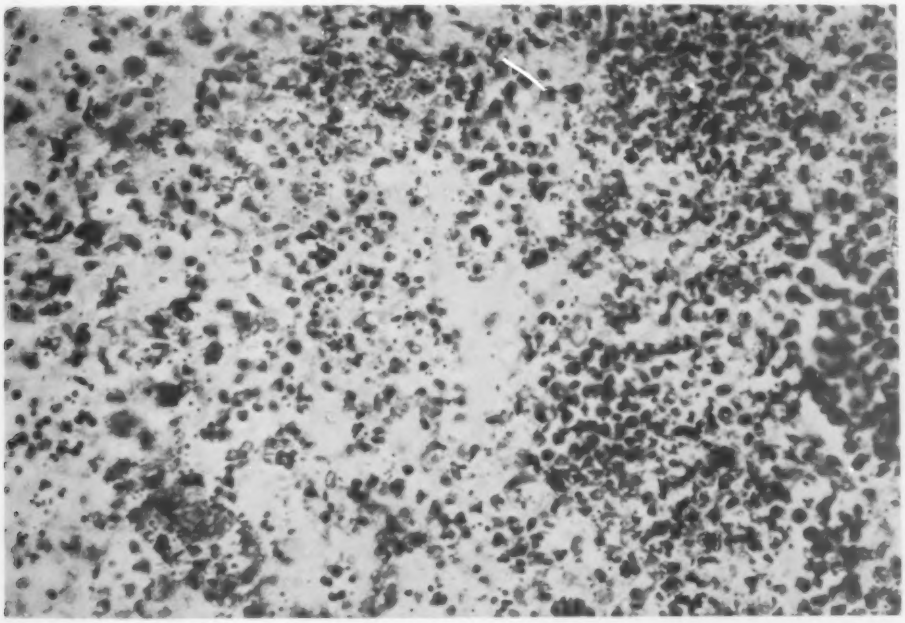

FIG. 4.—Dog No. 870. H. P. at location (B) of Fig. 3. Note leucocytic and lymphocytic infiltration with obliteration of alveolar walls.

increased in numbers since the three-hour stage, the former predominating four to one. A few large mononuclear giant cells were also present, some containing pigment.

*Silver Nitrate Cautery*—One-Day Specimen. *Gross Pathology.*—The surface of the lobe presented a huge solid, dark red area, a hæmorrhagic infarct. This consumed 50 to 75 per cent. of the entire lobe, with its apex located at the site of cauterization, at which point the pulmonary artery was found completely thrombosed. The necrosed bronchial wall and surrounding parenchyma at the apex of the infarct were beginning to form a sequestrum (Fig. 2).

*Microscopic Pathology.*—The bronchial wall and surrounding dead parenchyma were seen beginning to loosen from surrounding tissues. Marked leucocytic and lymphocytic infiltration (the former predominating) were present. Large mononuclear giant cells, some containing pigment, were noted throughout the pneumonic area (Figs. 3 and 4).

*Thermal Cautery*—One-Week Specimen. *Gross Pathology.*—The lobe surface was found deeply injected opposite the site of cauterization over an area of 1 by 4 centimetres. The bronchial wall was still in the process of sequestration at the site of injury. The surrounding parenchyma presented hæmorrhagic changes for a distance of 1 centimetre from the bronchial wall.

*Microscopic Pathology.*—The bronchial wall was still present but partly separated from the surrounding tissues. There was seen beginning regeneration of bronchial epithelium from around the cauterized area. The epithelium was a stratified type but very low, almost flattened, assimilating stratified squamous or transitional epithelium, and was found growing beneath the loosened bronchial wall. Beneath the regenerating epithelium was seen viable lung parenchyma heavily infiltrated with the pneumonic process described above. The leucocytes still predominated over the lymphocytes and the giant cells were more numerous than in the one-day stage. Also at this period there was beginning proliferation of fibroblasts, especially near the sloughing necrosed tissue (bronchial wall). In some areas a pneumonic exudate, consisting of leucocytes, red-blood cells and débris was interposed between the regenerating epithelium and lung parenchyma. This was well demonstrated by the elastic tissue stain.

*Silver Nitrate Cautery*—One-Week Specimen. *Gross Pathology.*—The lobe presented a large hæmorrhagic infarct consuming .8 to .9 of the entire lobe with its apex located at the site of cauterization. A bloody purulent discharge was noted coming from the bronchus. The bronchial wall and surrounding necrosed parenchyma may or may not have been completely sequestrated and cast-off by this time. The pulmonary artery was completely thrombosed.

*Microscopic Pathology.*—The bronchial wall and surrounding necrosed parenchyma, if present, were found partly separated from the surrounding viable lung tissue. Epithelial regeneration was similar to that found in thermal cautery of this stage. The fibroblastic proliferation beneath the epithelium was much more marked here than that found in the thermal cautery, as also was the pneumonic process, which extended farther from the necrosed bronchial wall. (The epithelium was the only part of the bronchial wall seen to regenerate where either type of cautery was used.) (Figs. 5 and 6.)

*Thermal Cautery*—Two-Week Specimen. *Gross Pathology.*—A somewhat pale and slightly elevated circular area about one inch in diameter was seen on the surface of the lobe opposite the site of cauterization. The bronchial lumen was somewhat constricted at the proximal edge of the injured area with dilatation distal to it, about 1.5 times the diameter of the bronchus, which was filled with a mucopurulent material (Fig. 7).

*Microscopic Pathology.*—The necrosed tissue had sloughed away. Fibroblastic proliferation was quite marked. Little evidence of hæmorrhage remained. There was more or less granulation tissue interposed between the parenchyma and the newly regenerated epithelium. Lymphocytes predominated over the polymorphs at this stage

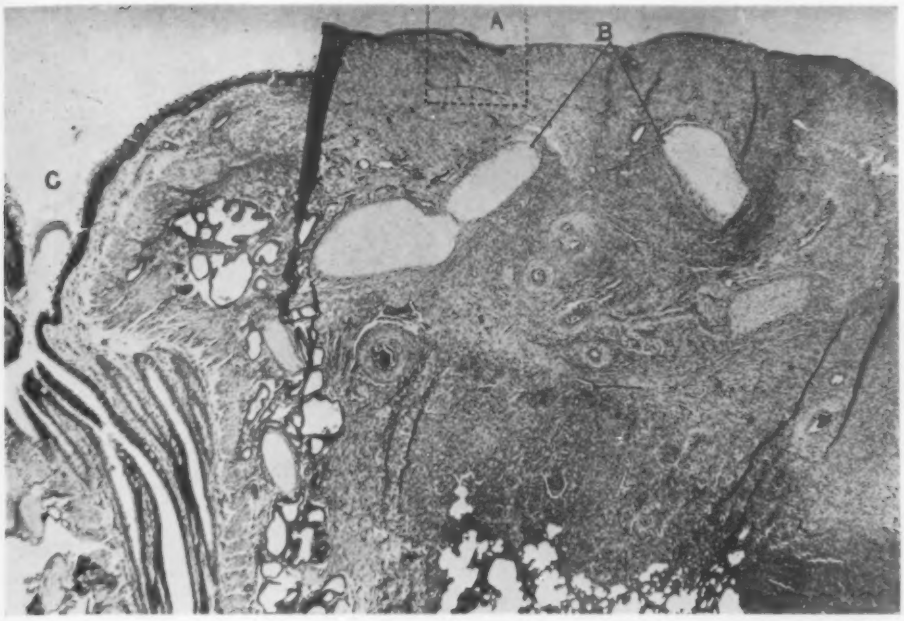


FIG. 5.—Dog No. 119A. Microscopic appearance of bronchial wall and surrounding parenchyma following silver nitrate cauterization, one week duration. Note (A) Regenerating epithelium. (B) Cartilages not cast off. (C) Uninjured bronchus. (x 35.)

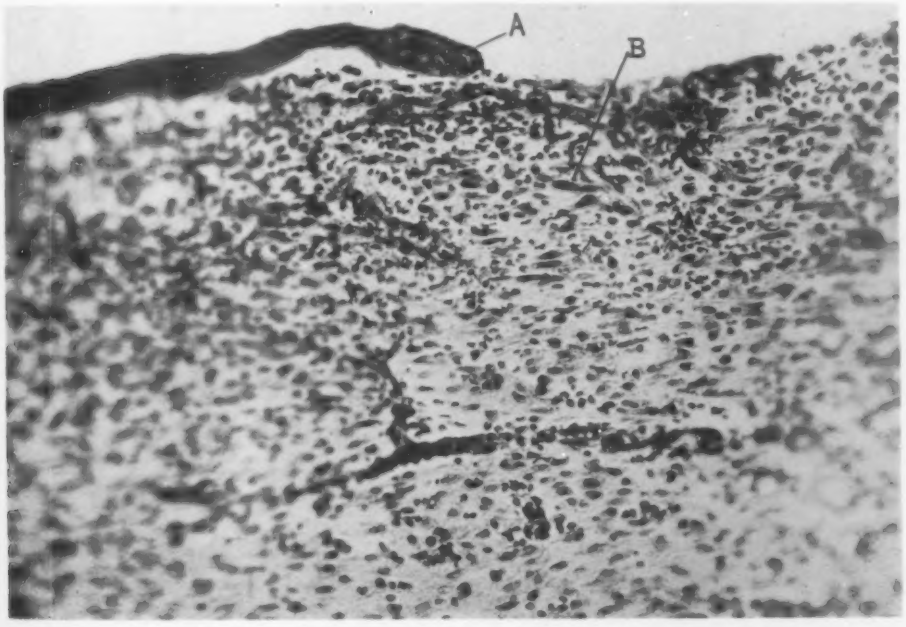


FIG. 6.—Dog No. 119A. H. P. at (A) Fig. 5. Note (A) Regenerating epithelium. (B) Fibroblasts.

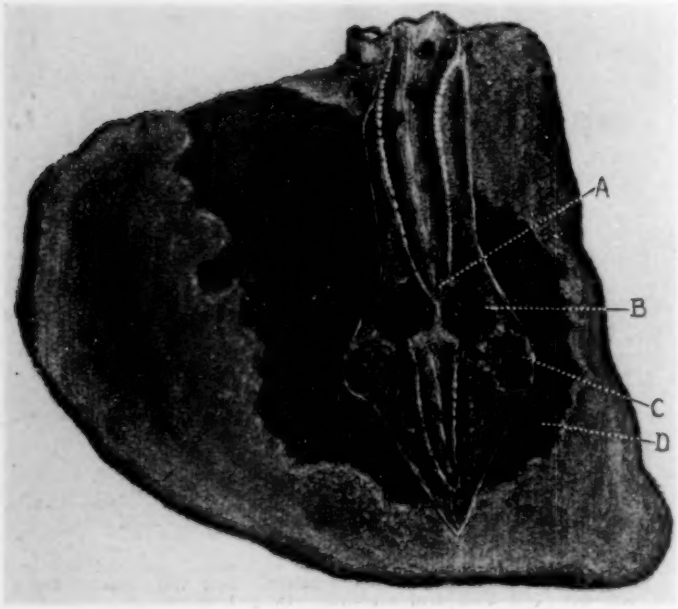


FIG. 7.—Dog No. 135A. Right lower lobe having received thermal cautery. Two days' duration. Note (A) Partial stenosis of bronchus. (B) Dilatation distal to stenosis with lack of cartilages. (C) Small abscess cavity communicating with dilatation. (D) Superficial fibrosis (not an infarct).


FIG. 8.—Dog No. 89A. Accessory lobe, one secondary bronchus of which received silver nitrate cautery, one-month duration. Note (A) Marked stenosis filled with a mucogelatinous substance. (C) Massive atelectasis of lobe distal to stenosis.

except where groups of polymorphs were noted here and there (small abscesses). The giant cells had increased in number.

*Silver Nitrate Cautery*—Two-Week Specimen. *Gross Pathology.*—A large hæmorrhagic infarct was still present. The bronchus was filled with a bloody discharge. The bronchial lumen was somewhat constricted at the proximal edge of the injured area, with a dilatation just beyond, 1.5 times the diameter of the bronchial lumen.

*Microscopic Pathology.*—The changes here were the same as those seen in thermal cautery at this stage. However, the fibroblastic proliferation was more pronounced and the process more extensive in the case of silver nitrate cauterization.

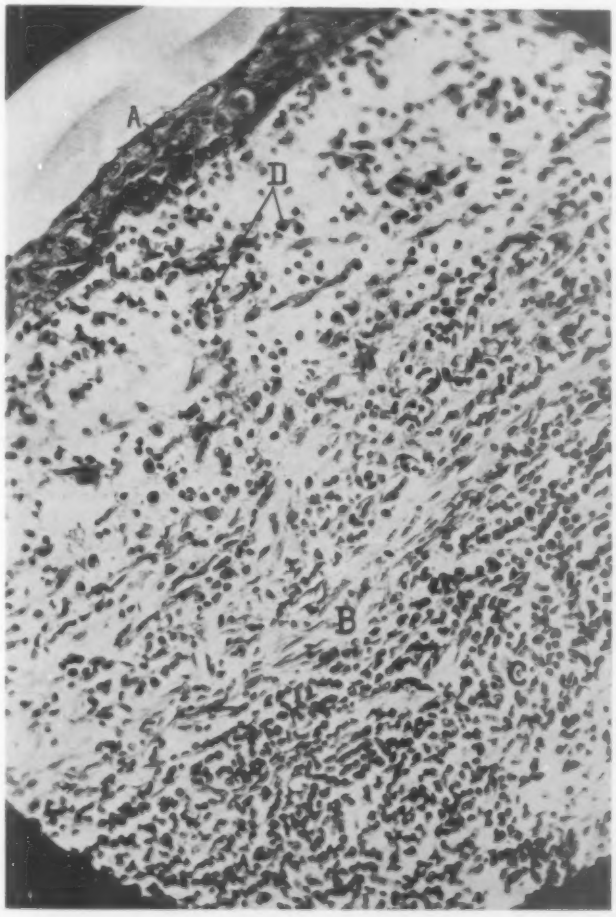


FIG. 9.—Dog No. 198A. Microscopic appearance following silver nitrate cauterization, three weeks' duration. Note (A) Regenerated epithelium. (B) Fibroblastic zone. (C) Lymphocytic zone. (D) Giant cells. Very few polymorphonuclear leucocytes present. (x 290.)

*Thermal Cautery*—Three to Four Weeks' Specimen. *Gross Pathology.*—The surface of the lobe appeared normal. The bronchial lumen was somewhat constricted at the proximal edge of the injured area with a dilatation distal to it filled with a mucogelatinous material. There was evidence of surrounding inflammation for a distance of 1 centimetre.

*Microscopic Pathology.*—The epithelium was completely regenerated. Surrounding fibrous tissue had replaced the other tissues of the bronchial wall. Lymphocytes and giant cells were still present.

*Silver Nitrate Cautery*—Three to Four Weeks' Specimen. *Gross Pathology.*—Two varieties of results were seen at this stage, *viz.*: 1. Much shrinkage and scarring of the lobe distal to the site of injury was seen. Bronchial stenosis was incomplete to complete. The pulmonary artery was thrombosed. 2. The surface of the lobe appeared almost normal. Bronchial stenosis was incomplete to complete. The lobe at times presented massive atelectasis distal to the stenosis. Larger air passages distal to the stenosis were dilated and filled with a dirty mucogelatinous material. The pulmonary artery was intact (Fig. 8).

*Microscopic Pathology.*—1. The epithelium was completely regenerated. Very much fibrous tissue surrounded the newly formed bronchial lumen and extended out to the lobe surface. Some round cells and giant cells were still present. 2. This differed from 1 in that the fibrous tissue extended for only 1 centimetre away from the bronchial wall and the alveoli distal to the stenosed bronchus were at times collapsed (Fig. 9).

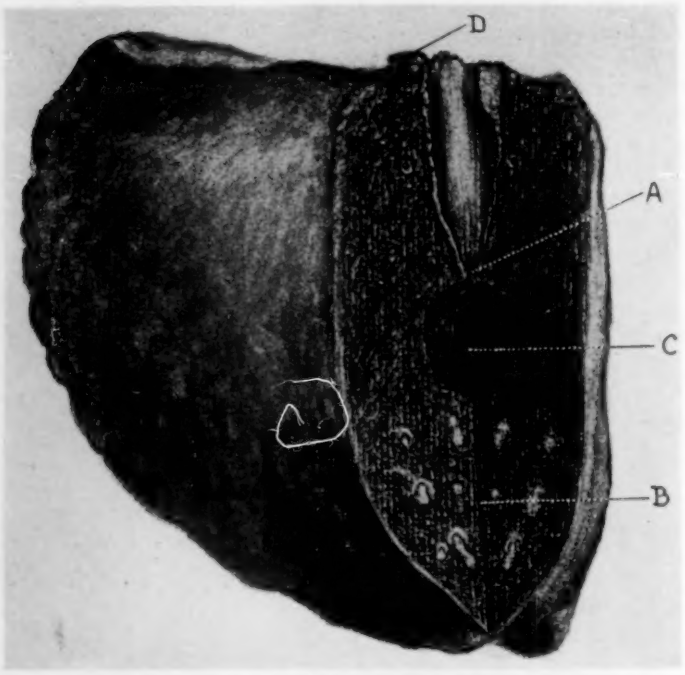


FIG. 10.—Dog No. 122A. Right lower lobe having received silver nitrate cautery, duration six weeks. Note (A) Complete stenosis of bronchus. (B) Normal air containing parenchyma but larger passages filled with mucogelatinous material. (C) Reorganizing area. (D) Pulmonary artery.

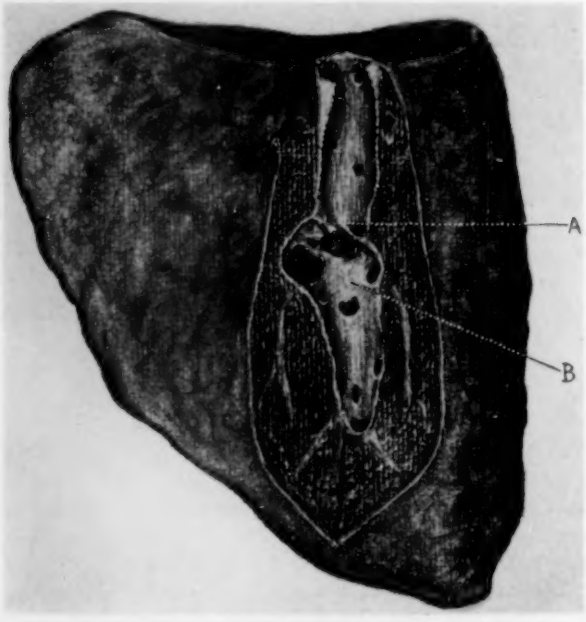


FIG. 11.—Dog No. 121A. Right lower lobe having received silver nitrate cautery, duration six weeks. Note (A) Partial stenosis of bronchus. (B) Dilatation distal to stenosis.

*Thermal Cautery*—Six-Week Specimen. *Gross Pathology.*—The surface of the lobe appeared normal. There was partial stenosis of the bronchus which presented a scarred appearance. (Lobe was somewhat shrunken in some cases.)

*Microscopic Pathology.*—Very little change was seen from that of the four-week stage. More fibrosis was present, but it was confined to the region immediately surrounding the bronchus. Giant cells and round cells were still present.

*Silver Nitrate Cautery*—Six-Week Specimen. *Gross Pathology.*—No. 1 of four-week stage. The lobe was much shrunken and was firm and rubbery in consistency. The pulmonary artery was thrombosed, the bronchus was completely stenosed. No. 2. The lobe surface appeared normal. Bronchial stenosis was incomplete to complete. Massive atelectasis was present at times distal to the stenosis. The larger air passages distal to the stenosis contained a dirty mucogelatinous material (Figs. 10 and 11).

*Microscopic Pathology.*—Little change from four-week stage was noted. The lymphocytes appeared to form a wall around the bronchus with much organizing fibrous tissue between it and the bronchial epithelium. In No. 1, the fibrous tissue extended out to the lobe surface. Giant cells were still present. (Fig. 12.)

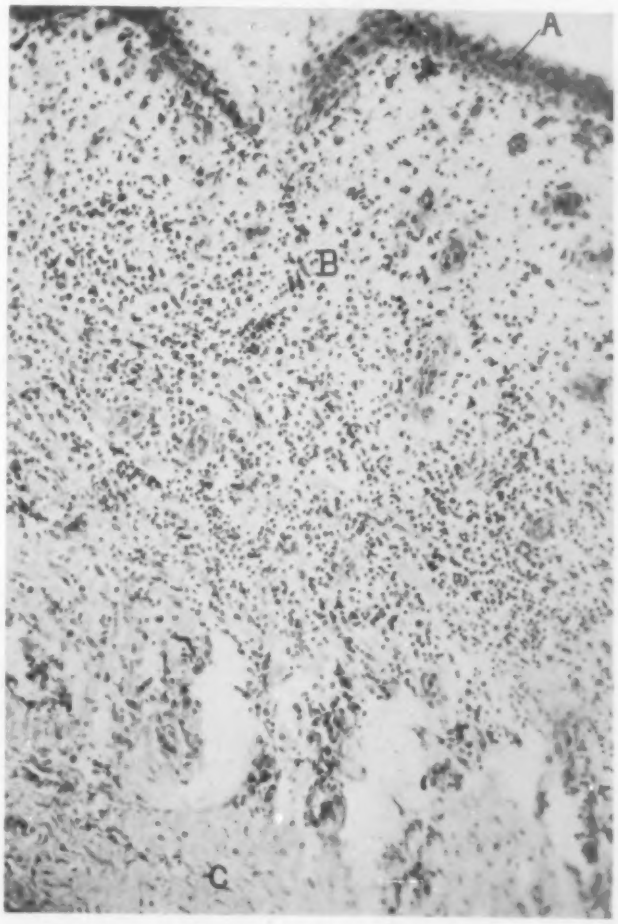


FIG. 12.—Dog No. 122A. Microscopic appearance following silver nitrate cauterization, duration six weeks. Note (A) Regenerated epithelium, (B) Reorganization, (C) Fibrous tissue. (x 200.)

*Thermal Cautery*—Eight-Week Specimen. *Gross Pathology.*—The surface of the lobe appeared normal. Bronchial stenosis was present, usually incomplete, at the proximal edge of the injured area, with dilatation distal to the stenosis. The pulmonary artery was intact.

*Microscopic Pathology.*—The entire bronchial wall was lacking except for the newly regenerated epithelium, which was a flat stratified type and lay on a fibrous tissue base.

*Silver Nitrate Cautery*—Eight-Week Specimen (two types of results). *Gross Pathology.*—1. The lobe was much shrunken and fibrotic. There was complete stenosis of the bronchus (Fig. 13). 2. The lung lobe appeared normal or atelectatic. Bronchial stenosis was incomplete to complete, with dilatation distal to the stenosis. The air passages beyond the stenosis usually contained a dirty mucogelatinous material.

*Microscopic Pathology.*—In both 1 and 2 the entire bronchial wall was lacking except for the regenerated epithelium, which was composed of several layers of flattened cells and lay on a fibrous tissue base. In 1 this fibrous tissue extended to the surface of the lobe (Fig. 14).

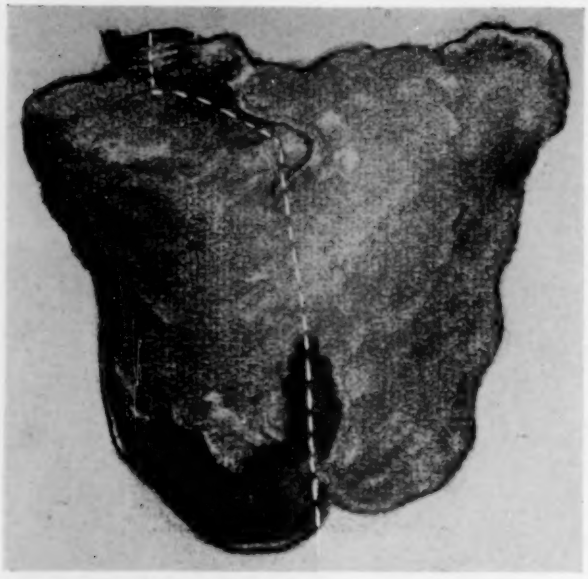

FIG. 13A.—Dog No. 896. Right lower lobe, two views, A and B. Silver nitrate cautery, duration two months.

*Repeated Thermal Cauterization.—Gross Pathology.*—The surface of the lobe appeared entirely normal. The bronchus was completely stenosed at the site of cauterization. Pulmonary vessels were intact.

*Microscopic Pathology.*—The bronchial wall at the site of injury was composed of white fibrous connective tissue with pieces of dead cartilage scattered throughout. A fibrous connective tissue septum, lined on either side by a low stratified epithelium, stretched across the bronchial lumen, totally obstructing the passage of air (Fig. 15).

*Discussion.*—The results given above are those which usually follow this type of injury. However it is readily understood how variations may occur with slight changes in the fundamental factors, *i.e.*, in one dog a small abscess formed just beneath the surface of the lobe and communicated with the bronchial dilatation at the site of injury as the result of liquefaction and sloughing rather than reorganization. Another dog's lung showed the pulmonary artery wall adjacent to the cauterized bronchus to consist of a thin partition of granulation tissue stretched between the bronchus and the artery, the arterial lumen being intact. A third, and more commonly seen variation, was massive atelectasis in the lobe, distal to the partially stenosed bronchus. Our view

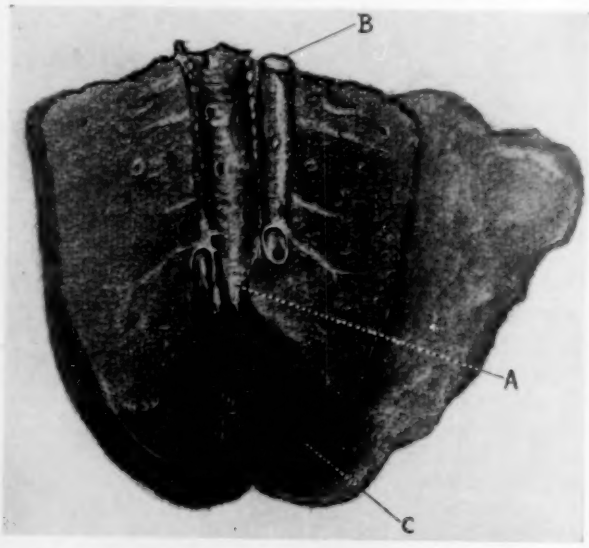


FIG. 13B.—Note (A) Complete stenosis of bronchus. (B) Pulmonary artery. (C) Fibrous tissue. Slow organization in centre.

concerning the mechanism of production of this collapse is given in another publication.[14]

The reparative processes following an injury to a bronchus depend upon the severity of the injury, both to the bronchial wall and to the vascular supply of the lung lobe. It is to be noted that the damaging agent caused a complete necrosis of the bronchial wall, with regeneration of only the epithelial lining. This failure of the bronchial wall, other than the epithelium, to regenerate, may explain the cause of failure to obtain a satisfactory closure of the stump in lobectomies and pneumectomies where little or no peribronchial tissue covers the stump (as suggested by Bettman).

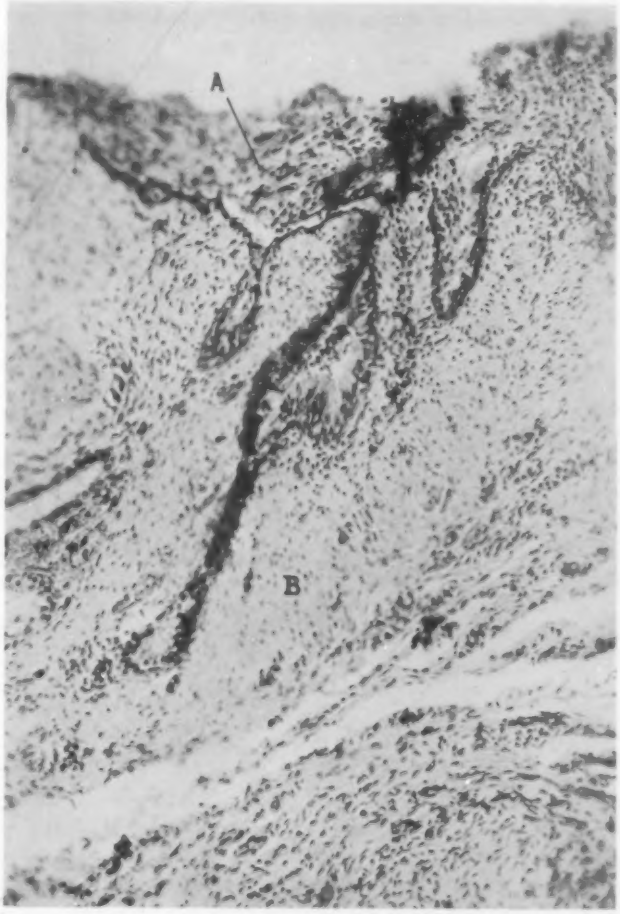


FIG. 14.—Dog No. 896. Microscopic appearance following silver nitrate cautery, duration two months. (A) Regenerated epithelium. (B) Fibrosis. (x 200.)

In the case of silver nitrate cautery, the adjacent peribronchial parenchyma was also destroyed and cast off. The resulting stenosis of the bronchus occurred at the proximal end of the injured area, while the accompanying dilatation was just distal to the stenosis and in the region of greatest destruction. This leads one to believe it was a passive rather than an active dilatation.

As to blood-vessel damage, thermal cautery never produced thrombosis of the pulmonary artery, while silver nitrate was always followed by thrombosis and hæmorrhagic infarct formation within twenty-four hours. In later stages, it is to be noted, the infarcted area either became fibrosed or resolved into fairly normal air-containing parenchyma, depending upon the restoration of the vascular supply; perhaps both time and degree of revascularization. At times there was a piling up of organizing pneumonic exudate, which was lined by the

regenerating epithelium, thus forming polyps. This was noted by Winternitz [15] in his work with hydrochloric acid insufflation in rabbits. He also found the epithelium to regenerate very rapidly, at times forming inclusions extending beneath the surface of the air-passage wall.

The rapidity of the regeneration of the lining epithelium, together with the lack of regeneration of the other tissues of the bronchial wall, may throw some light on the cause of failure of many persistent bronchial fistulas to respond to therapeutic measures. Work is being carried out on this problem at the present time, which will appear in a subsequent publication.

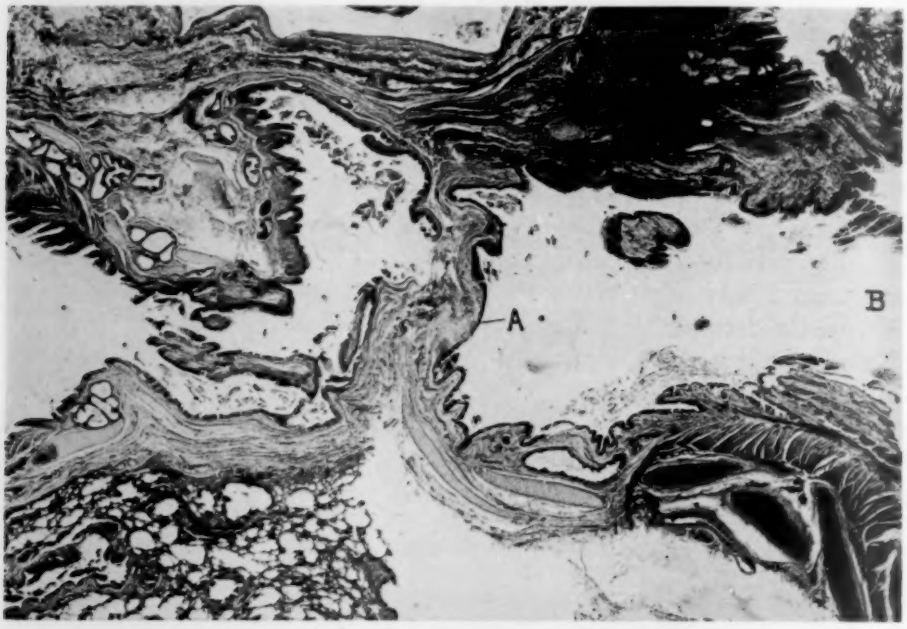


FIG. 15.—Dog No. 935. Microscopic appearance following repeated thermal cauterization over period of approximately six months. (A) Fibrous tissue septum lined by epithelium producing complete obstruction. (B) Origin of bronchus. (x 25.)

A brief summary of the comparison of the two types of cautery follows:

| | Thermal bronchial wall necrosis | Silver nitrate bronchial wall necrosis; adjacent parenchymal necrosis; blood-vessel damage |
|---|---|---|
| 1. Degree of injury | | |
| 2. Thrombosis of pulmonary artery | never | always |
| 3. Degree of early reaction | moderate | marked |
| 4. Infarct formation | never | always |
| 5. Regeneration of epithelium | early | early |
| 6. Regeneration of bronchial wall other than epithelium | none | none |
| 7. Degree of fibroblastic reaction | moderate | marked |

Repeated thermal cauterization resulted in complete stenosis of the bronchial lumen. Although only two dogs were treated in this manner, the results were the same in both.

The production of stenosis was a very gradual process which, in a bronchus with a lumen of .4 inches in diameter, extended over a period of approximately six months. Two factors were concerned in this process, *viz.*: the production of granulation tissue within the bronchial lumen; and fibroblastic proliferation around the bronchial wall with fibrous tissue formation.

In view of these findings it is logical to believe that persistent bronchial fistulæ may be permanently closed by means of repeated thermal cauterization.

## CONCLUSIONS

1. Silver nitrate and thermal cautery may produce very similar lesions and stimulate very similar reactions when applied to a bronchus.

2. An injury due to one of the above agents, sufficient to destroy the entire bronchial wall, may produce the following changes:

*a*. Sequestration and sloughing of the entire thickness of bronchial wall in one to two weeks.

*b*. Hæmorrhagic infarction within twenty-four hours after the use of silver nitrate.

*c*. A pneumonic process which either resolves or goes on to various degrees of fibrosis.

*d*. Regeneration of a low stratified epithelium within six days (before sequestrated wall has sloughed away).

*e*. No regeneration of bronchial wall other than epithelium.

*f*. Much fibrous tissue formation at site of cautery.

*g*. Stenosis of bronchial lumen, incomplete to complete, accompanied by dilatation of the bronchial lumen just distal to the stenosis.

*h*. Air passages distal to a completely stenosed bronchial lumen filled with a dirty mucogelatinous material.

3. Repeated thermal cauterization results in complete stenosis of the bronchial lumen, the process extending over a period of months depending upon the size of the bronchus.

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# MORTALITY AND END RESULTS OF OPERATION FOR ABSCESS OF THE LUNG*

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The treatment of lung abscess has been considered in recent years so carefully, and from so many different angles that we are in a fair way to a better understanding of the many complicating factors concerned. It is essential for the surgeon to understand the etiologic factors responsible for the abscess and particularly the possibility of foreign body obstruction of a bronchus, the pathologic nature of the process, the extent of associated bronchiectasis, the presence of multiple cavities, the existence of a gangrenous wall, etc., if he wishes to obtain the best results from treatment.

Recognizing that drainage of the collection is the means by which cure is obtained and that a residual bronchiectasis may be responsible for recurrence, it is apparent that either the bronchial tubes, or an external opening may be used for drainage and that compression or removal of the affected part of the lung may be necessary to prevent recurrence. As this paper is concerned only with the results of external drainage most of the details regarding other methods of treatment will not be considered.

In any case of lung abscess, rest in bed, postural drainage, and bronchoscopic aspiration must be considered as primary treatment. Some patients undoubtedly recover spontaneously and Lord (Boston M. and S. Jour., 1927, vol. cxcvii, p. 333) estimates this as 10 per cent. Graham (Archives Surg., 1927, vol. lxxxvi, p. 174) says that in his experience, 25 per cent. or more heal spontaneously. Posture undoubtedly favors drainage of the abscess and in favorable cases the approximation of the granulating walls permit healing. Lord believes that if the disease has lasted eight weeks or less, the chances of recovery are about 20 per cent., but if the process has lasted a longer time than this there is little to be expected from palliative measures.

Bronchoscopic aspiration has proved its worth. With due consideration of the spontaneous cure of many cases, and the effect of rest and postural drainage there is no doubt but that additional relief can be given by the bronchoscope. Eliminating those cases of suppuration associated with foreign body in the bronchus which always indicate bronchoscopy, the presence of pus in the bronchi in a case of proved lung abscess usually means that some drainage is occurring. The bronchoscope acts by removing heavy plugs of mucus or pus, thus providing better drainage. Kramer (Surg. Clinics North America, 1928, vol. viii, p. 377) reports 27 per cent. of cures in 105 patients,

* Read before the Philadelphia Academy of Surgery, December 2, 1929.

observed for a period of six months or more. Kernan (Arch. Surg., 1928, vol. xvi, p. 215) considers 45.5 per cent. of 68 patients as cured by bronchoscopy and Clerf (Atlantic Med. J., 1928, vol. xxxi, p. 911) reports 51 per cent. of cures after bronchoscopic treatment of lung abscess following tonsillectomy. Clerf also noted that 79 per cent. of his patients were cured in whom the treatment was instituted within three months after the onset of symptoms.

The status of artificial pneumothorax is not clear. I would judge that it might be worth-while in the occasional patient with a non-adherent lung, a single cavity, especially one near the hilum, and a free bronchial outlet. It has possibilities of great danger in peripheral abscess from rupture of adhesions. Arsphenamine is useful in cases with fetid sputum and when spirochetes are present in the sputum.

External drainage will be found necessary in about 50 per cent. of patients suffering from lung abscess, although in clinics where expert broncho-

TABLE I

*Fatal Cases*

| Etiology | Age | Location | Duration | Death |
|---|---|---|---|---|
| 1. Tonsil | 25 | R.L. | 10 weeks | Sudden collapse. 12 days |
| 2. Pneumonia | 36 | R.U. | 14 months | Abscess other lung. 2 days |
| 3. Pneumonia | 75 | R.U. | 6 months | Gang. extremities. 23 days |
| 4. Pneumonia | 51 | R.L. | 6 weeks | Pneumonia. 17 days |
| 5. Broncho. Pneumonia | 47 | R.U. | 1 month | Abscess other lung. 37 days |
| 6. Pneumonia | 63 | R.U. | 2 weeks | Abscess other lung. 2 months |
| 7. Tonsil | 9 | R.L. | 5 months | Cerebral abscess. 1 month |
| 8. Broncho. Pneumonia. | 67 | R.U. | 6 weeks | Pyo-pneumothorax |
| 9. Broncho. Pneumonia. | 46 | R.U. | 3 months | Cerebral abscess. 12 days |
| 10. Tonsil | 54 | R.U. | 1 month | Abscess other lung. 1 month |

scopic treatment is given, this number may be lessened if there are many of the post-tonsillectomy cases. Generally we should advise operation in cases of peripheral abscess with pleural involvement or with empyema, in multiple abscess, or lobar bronchiectasis, and after a trial of several months of postural drainage supplemented by bronchoscopic aspiration. I also believe that in those cases of well localized single abscess with good surrounding lung, external drainage is safe and effects a more rapid cure. Each patient must be treated as an individual and treatment advised after careful study and a preliminary bronchoscopy.

*Mortality.*—Most clinicians and bronchoscopists take a pessimistic attitude towards operation and no doubt this has been brought about by the high mortality attending it. There are many series of cases on record, but a review of the literature indicates an average mortality of 35 per cent. The incidence of death from operation depends upon the condition of the patient and the proper selection of the time for operation. During a period of seven years (1922–1928) thirty-five patients have been referred to my service in the Hospital of the University of Pennsylvania for external drainage of a

lung abscess. Practically all had been under conservative treatment previously and many treated by bronchoscopic aspiration. There were ten deaths (28.5 per cent.) but the subjoined table indicates the serious nature of the infection in these cases.

Four patients had involvement of both lungs and in one, nothing was done except the preliminary stage of rib removal under local anæsthesia. Two patients died of cerebral embolus and one of these was apparently well and up in a wheel chair when stricken with the hemiplegia. He was operated on by Doctor Frazier but succumbed. Another patient was an old woman of seventy-five years. She was a poor risk and probably should not have been operated on; pulmonary œdema and toe gangrene were terminal affections. Seven of these patients, therefore, were nearly hopelessly handicapped.

TABLE II

*Age Relation to Mortality*

| | All Cases | Tonsil Group | Others |
|---|---|---|---|
| Recovery | 35 | 13 | 22 |
| Deaths | 10 | 3 | 7 |
| Per cent. | 28.5 | 23 | 32 |
| Total average age | 36 years | 29 years | 44 years |
| Fatal cases | 47 " | 29 " | 55 " |

| | Cases | Deaths | Mortality |
|---|---|---|---|
| Average age, 36.3 years | | | |
| 36 years or less | 19 | 3 | 16 per cent. |
| Over 36 years | 16 | 7 | 44 " " |
| Mean age, 35 years | | | |
| Below 35 years | 17 | 2 | 12 " " |
| Over 35 years | 17 | 8 | 47 " " |

Another patient was operated upon seven months after onset of symptoms following tonsillectomy. The operation was done in two stages and the abscess easily drained. Eight days later she suddenly developed symptoms of shock and died in twenty-four hours. No post-mortem was obtained but the symptoms were not cerebral. The pulse was always very rapid. The operation was done in 1922 and ether anæsthesia was used for the first stage and the lung sutured to the parietal peritoneum. Both of these procedures were abandoned that year. The ninth death occurred in a patient who had a large abscess in the lower right lobe. Apparently the operation was properly conducted and for six days after drainage the temperature was normal and the pulse low. Signs of a spreading lung involvement then appeared with death three days later. Autopsy refused. The tenth death was due to bad management. The patient was sixty-seven years old and quite ill. He was operated upon too soon. The abscess was not properly located and the

pleura opened during the evacuation of the abscess. Later he developed a pyo-pneumothorax and died from this because at autopsy the abscess was found perfectly drained. Therefore, in a review of this list we feel that in two cases death might have been avoided, but that in the others we were helpless to prevent it.

I have been struck by another factor in studying mortality and that is the influence of age. Lilienthal (Arch. Surg., 1928, vol. xvi, p. 206) mentions this, the mortality in his series being 42 per cent. in those under fifty and 63 per cent. in those above that level. In this series the mortality of operation in 27 patients under fifty years was 18.5 per cent., whereas in eight over fifty it was 62.5 per cent.

*End Results.*—Careful studies of the end results of operation are rarely noted in the literature. In a series of 100 cases reported by Miller and Lambert (Amer. J. Med. Sc., 1926, vol. clxxi, p. 81) 47 were operated upon with 19 death and 20 cures (42.5 per cent.). In the group of cases reported in this paper there are 14 cures (40 per cent.). By this is meant that the present condition of every patient has been obtained and many have been personally examined, all at least one year since operation.

TABLE III

*End Results*

| |
|---|
| 25 patients survived operation |
| 14 are cured. 40 per cent. of total. 56 per cent. of survivals |
| 7 patients show varying grades of improvement |
| 4 later deaths. Final mortality 40 per cent. |

In the cured group the patients have been operated upon a period of one year to six years and seven months. They now have no cough or expectoration, no sinus and are able to work. Most have been checked by röntgen examination. The trunk shadows are usually increased, the diaphragm usually high and fibrosis is suggested.

In the "improved" group two patients seem cured of the abscess. Both have been operated upon over six years but they still speak of cough and some expectoration at times and neither is able to do much work. One of these patients, forty-five years old at the time of operation, had had an unproductive winter cough for twenty-two years. He was operated upon in May, 1923, and now in November, 1929, still has cough and some râles, but is otherwise well. The other, fifty-four years old, was also troubled with cough for years. He was operated upon in October, 1922, still has cough and expectoration, some myocardial disease and attacks of dyspnœa. From the surgical point of view these patients are cured, but clinically are not well.

In a third patient a left lower lobe abscess was complicated by a bilateral lesion and demonstrated tuberculosis. The abscess was drained. Two years later he is markedly improved, but is in a sanatorium. The abscess on the

side operated has healed. A fourth patient developed abscess after pneumonia and was operated upon one year later. Drainage was followed by a cautery operation. About fourteen months later the sinus has healed; there is still cough and slight expectoration; there is no fever and hemoptysis, and while X-ray shows a small cavity the appearance is that of great improvement.

The other three patients are still suffering from suppuration in the lungs. They have drainage, cough and expectoration, attacks of fever and X-ray evidence of multiple cavitation. All have had cautery operations, but are in need of further surgical treatment. They are in no immediate danger, but probably, sooner or later, will die from the lung affection. Two of these patients had the first operation eighteen months ago, and in one, a year has elapsed.

Finally, regarding those patients who succumbed after discharge from the hospital.

TABLE IV

*End Results*

| | All Cases | Tonsil Group | Others |
|---|---|---|---|
| Final mortality | 40 per cent. | 31 per cent. | 45.5 per cent. |
| "Improved" | 20 per cent. | 15 per cent. | 22.5 per cent. |
| Cured | 40 per cent. | 54 per cent. | 32 per cent. |

One patient was a man, thirty-seven years of age, who had pneumonia ten weeks before admission. He came in with symptoms and signs of abscess. This was easily evacuated and the patient made a slow recovery. However, he had a bronchial fistula and was re-examined in December, 1922, at which time he had some cough and expectoration. We heard nothing further until this follow-up was begun when it was reported that he had died in January, 1923, eleven months after operation, from "pneumonia." Undoubtedly his death resulted from a continuance of the original disease.

The second case was a man of sixty, whose lesion had existed five months, following a chronic cough of many years. I evacuated the abscess in the left upper lobe and at the time suspected malignancy of the lung. He had a bronchial fistula at the time of drainage and six months later died, his physician stating that the drainage had continued and that "death was not unlike a case of pulmonary tuberculosis."

A third patient developed an abscess after tonsillectomy and was admitted to the hospital thirteen months later. An abscess in the left upper lobe was opened, a secondary pocket subsequently cauterized, and later she seemed in fairly good condition and was sent home. She then went West, but her physician writes that she died from continued trouble in the lung about a year after the original operation.

The fourth patient was operated upon for a left lower lobe abscess following pneumonia. Three months later there was evidence of a suppurative condition in the left upper and right lower lobes. Bronchoscopic treatment was given, but during an intermission the patient died suddenly from cerebral manifestations. No autopsy was obtained.

A fifth patient was operated upon in September, 1926, for a right upper lobe abscess following tonsillectomy. He was sixty-three years of age. He seemed to have done well for a while, but died in March, 1928, from a cause unknown.

These five patients and the three patients who are hanging on in a slightly improved or unimproved condition, constitute a group of patients

difficult to treat. Lilienthal states that, "if recovery is not complete, whether or not operation has been performed, the possibility is that the patient will finally succumb to his disease or to its sequelæ," as he well says, "one must not be deceived by discharge of a living patient from the hospital."

After external drainage has given relief from sepsis and cough and expectoration has lessened, a careful review with the X-ray is necessary to exclude the existence of multiple cavity. Sometimes this can be determined in the wound with a light. If such exists the surgeon must plan an early cautery operation after the method of Graham. Only by breaking down all pockets can we expect to cure this group, otherwise they leave the hospital "improved," only to suffer from a recurrence. During four years Graham (Arch. Surg., 1929, vol. xviii, p. 531) operated on 45 patients by this method, of which 24 per cent. died later from various causes, the hospital mortality being 6.6 per cent.

This paper would be incomplete without mention of lobectomy. It may be that we will find this the best procedure in cases of multiple cavity and limited bronchiectasis. During the period covered by this paper I had not performed lobectomy for pulmonary suppuration.

## CONCLUSIONS

1. Lung abscess is a serious disease and the end result of a group of patients can only be determined a long time after treatment.

2. External drainage is indicated (1) after failure of conservative measures, (2) in peripheral abscess with pleural involvement (3) in multiple abscess.

3. Cautery excision or lobectomy is indicated in (1) multiple abscess and (2) in abscess with lobar bronchiectasis.

4. A series of cases is reported with an operative mortality of 28.5 per cent. and a final mortality of 40 per cent. Fifty-six per cent. of the survivors are clinically well. Two patients recovered from the abscess, but still have the cough they had for many years, giving 64 per cent. of satisfactory results in the survivors.

5. Those patients who survive the operation, but who do not get permanently well will be likely to succumb to the effects of the disease. Perhaps an increased use of the cautery method of extirpation of the diseased area, done early, may result in a greater percentage of ultimate cures, even though the immediate mortality may be higher.

# DIAPHRAGMATIC HERNIA

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A REVIEW OF THE LITERATURE AND ETIOLOGY OF THE CONDITION, WITH A REPORT OF FOUR PERSONAL CASES

Diaphragmatic hernia has been largely in evidence in both American and foreign medical literature the last few years. Sisk[1] computes that about one thousand cases have been published since 1920. Nevertheless, this condition wears such a number of faces, both clinically and on the operating table, that something may still be learned from each new case reported in full. This point was recently urged by Haberer,[2] who refused to preface his case history with an apology and deplores the possibility of any such case being lost in statistical summaries before it has been published in detail.

Prior to the use of the röntgen ray, a diagnosis of diaphragmatic hernia was made in only one or two instances before the opening of the body, but in the last few years the condition has been correctly diagnosed in a number of cases on clinical grounds alone and even by the general practitioner.[3] This advance in diagnostic feasibility has been made possible by the many complete and thoughtful case reports that have been published, in which the symptoms, bewildering in their variations from case to case, have been interpreted in the light of the röntgenogram and screen examination and by the details of anatomic and functional derangement exposed at operation or autopsy.

As we become more familiar with this condition, we find that it is not as rare as was formerly supposed. From a surprise of the operating room or a curiosity of the autopsy report, it has become a possibility to be considered, routinely, in every case with unexplained symptoms referable to the heart or lungs or to the digestive system. Importance should not be attached to the fact that the symptoms have begun in adult life with no trauma in the recent history. Diaphragmatic hernia of congenital origin may become manifest at any age, and when trauma is the cause years may elapse between the injury and the onset of symptoms of the hernia.

The cause of congenital diaphragmatic hernia is the failure of the component parts of the diaphragm, growing out toward each other, to meet and unite. If this developmental failure occurs before the membranous diaphragm is complete, the defect will be total and the thoracic and abdominal cavities will be in free communication. The hernia will be without a sac—a so-called "false" hernia. If, on the other hand, it occurs later, during the formation of the muscular diaphragm, a layer of apposed pleura and peritoneum will separate the cavities and will form a sac for the ascending abdominal viscera. In the first type, it is probable that the herniation of the organs takes place during intra-uterine life, even when symptoms do not appear till late. The viscera acquire tolerance for their crowded condition and,

since they can move more freely, there is less danger of strangulation or kinking than when they are confined in a sac. In the second case, the herniation may not take place till long after birth. Indeed, if the defect is small, it may never take place. The diaphragm contains a weak spot and under some unusual condition of internal tension the hernia may at any time occur or it may develop gradually. Thus it is usual to consider all cases of diaphragmatic hernia in which there is no history of a penetrating wound or of external violence to the lower thorax or upper abdomen as of congenital origin, but the term "acquired" hernia, used by Hedblom[4] is, perhaps, preferable for hernias that take place slowly, at any time after birth, through an anatomically weak area, such as the œsophageal hiatus or the foramen of Morgagni.

Infants with large defects of the diaphragm do not, as a rule, survive for many hours, but Hedblom[4] mentions two cases in which the patient reached adult age with congenital absence of half of the diaphragm. Kleine[5] last year reported what he believes is the most extensive congenital diaphragmatic hernia thus far recorded. The stomach, duodenum, entire small intestine, ascending, transverse, and part of the descending colon, the pancreas, spleen, and upper pole of the left kidney, and the left suprarenal gland were all in the left pleural cavity. The opening in the diaphragm extended from the œsophageal hiatus to the left wall of the abdomen, but it was not in connection with the natural apertures for the passage of the œsophagus, vena cava and aorta. From right to left it measured 4.7 centimetres, the sagittal diameter was 3.3 centimetres. The sigmoid colon was constricted in the shape of an hour-glass where it passed through the diaphragm, but the lumen was not closed; it contained meconium. The child was born after a normal labor and was not otherwise malformed. Immediately after birth it breathed and cried, but respiration soon became superficial and irregular and after four hours respiration ceased.

Kleine does not believe that the hernial opening in this case was due to defective development of the diaphragm, but holds that an exaggerated ascension energy of the left kidney was the responsible factor. Normally, the kidney reaches its final position under the diaphragm in the fifth to the sixth week of embryonic life. The pleuroperitoneal foramen does not close before the eighth or ninth week. If the impulse that causes the ascent of the kidney should be abnormally strong, that organ could, therefore, push up beyond the diaphragmatic level at this point and prevent closure of the temporary physiologic aperture. Later this opening could become enlarged and admit other abdominal organs. This is the theory advanced by von Mikulicz-Radecki[6] to explain a case of right-sided renal dystopia with true diaphragmatic hernia. Gruber[7] and Liepmann[8] published similar cases but did not suggest this etiology. A fact which Kleine believes supports this theory of origin for his case is that the kidney was firmly affixed to its bed, whereas the other herniated organs were free. The extreme grade of the hernia is accounted for by the persistence of the common mesentery.

Hernia, congenital or traumatic, is much more frequent through the left half of the diaphragm than through the right, which, in addition to being, it appears, stronger, is protected by the presence of the liver. The greater interest, therefore, attaches to Jansen's[9] case, in which almost the entire right leaf was absent. A diagnosis of diaphragmatic hernia was made by means of the röntgen ray and a small amount of ingested barium on the sixth day of life. The child lived three weeks. At autopsy the entire small intestine and part of the large intestine were in the right thorax.

Schwartz [10] reports a case of congenital absence of all that part of the diaphragm that takes its origin from the quadratus lumborum and the left pillar. Since there was a hernial sac formed apparently of apposed pleura and peritoneum, the anomaly dated from the fetal rather than the embryonic period. The œsophagus passed into the opening and was found, together with the right pneumogastric nerve, in the wall of the sac. This hernia did not reveal itself for forty-seven years.

A slightly different cause for congenital hernia is congenital enlargement of one of the natural permanent openings in the diaphragm. Of these, the opening for the passage of the œsophagus is the one that most often gives rise to the condition. Barsony [11] thinks that these hernias are not uncommon, but adds that their clinical importance is uncertain. They may be caused by a congenitally short œsophagus; in this case, a portion of the stomach, beginning with the cardia, will be above the diaphragm and reposition is impossible. The opaque substance runs promptly into the subdiaphragmatic portion of the stomach and, with the patient standing, röntgen examination may yield no evidence of hernia; with the patient in the prone position, the herniated portion of the stomach may simulate dilatation or diverticulum of the œsophagus. In other cases, the œsophagus, though of normal length, has not entered the abdomen, and the hernia is situated beside or behind it. Or, again, the œsophagus is not fixed beneath the diaphragm and ascends, taking the stomach with it through an enlarged hiatus. Schilling [12] shows röntgen pictures in a case in which the entire stomach, together with the first part of the duodenum, had ascended through the œsophageal hiatus. The patient was a man of fifty-eight with no history of trauma. Symptoms, chiefly pain over the sternum, appeared first at the age of twenty-seven. At no time were there symptoms from the side of the lungs or heart.

Failure of the œsaphagus to attain its adult length, on account of cicatrices resulting from the swallowing of a corrosive substance at the age of twelve years, which necessitated treatment with bougies over a period of many years, is offered by Samuelsen,[13] in explanation of an œsophageal hiatus hernia. The gastric symptoms began at the age of fifty-eight.

The œsophageal opening may become enlarged as the result of trauma. Sisk's [1] patient fell a distance of 14 feet, landing on head and shoulders. No special injury was noted at the time, but, soon after, the man began to have pain and a feeling of fullness in the lower part of the left chest, with attacks of nausea and occasional vomiting; lying in certain positions in bed caused distress. A year later he had a severe attack, which was diagnosed cholecystitis (an error that has been made in a number of cases of diaphragmatic hernia). It was not until five years after the fall that a diagnosis of an intermittent hernia through an enlarged œsophageal hiatus was made röntgenologically.

Most writers on this subject include diaphragmatic hernias from falls and accidents involving crushing of the trunk under traumatic hernias, but some are of the opinion that such accidents never cause a rent in a wholly

intact diaphragm and that they result in hernia only where there is an already existing congenital weakness or defect of the muscle. A number of diaphragmatic hernias have been reported following automobile accidents, where a person has been pinned under a car with compression of the lower thorax and the epigastrium, and they have been explained by rupture of the diaphragm on sudden increase of the intra-abdominal pressure.

Lafourcade [14] offers a different explanation for his case. The tear in the diaphragm started at the spinal column and passed in a forward and slightly lateral direction to the neighborhood of the internal surface of the ninth rib. It corresponded to the site of the juncture of the left leaf of the phrenic centre with the muscle fibres of the diaphragm. While admitting that this rent could have been caused directly by increased intra-abdominal pressure, he thinks it probable that this pressure widened the base of the chest, separating the costal insertions from the phrenic centre insertions of the muscle fibres of the diaphragm, and causing rupture of the latter at this point. The patient was a man, aged thirty-three, who, in a collision, had been struck by a part of an automobile in such a way that the epigastrium and lower part of the thorax were forcibly compressed anteroposteriorly.

Lecene's [15] patient, a vigorous, muscular man of twenty-five, sustained a rent in the diaphragm extending from the œsophageal orifice to the middle of the phrenic centre—about 15 centimetres from being pinned under an overturned automobile, with compression at the "waist" level for a number of minutes. There were no rib fractures and the result seemed to be limited to shock, but when, after a week, he was given semi-solid and solid food, he vomited it all, usually at once. The taking of liquids was followed by palpitation of the heart. A month after the accident, röntgenologic examination with barium showed the stomach almost wholly in the left half of the thorax.

Sauerbruch [16] mentions that crushing is more likely to result in diaphragmatic hernia in young persons than in those of more advanced age, because of the elasticity of the thorax in youth, which permits of its being widened and the diaphragm, in consequence, stretched.

The most frequent causes of frankly traumatic hernia are gunshot, shell or stab wounds of the diaphragm. Since such wounds commonly involve all of the layers of the diaphragm, these hernias seldom have a sac. It is probable that the hernia (more correctly, prolapse) follows immediately on the injury in such cases, but the length of time that has in many instances elapsed between the injury and the appearance of any symptoms of consequence from the side of the hernia is certainly remarkable—ten years in Lecene's [17] case of shell injury of the left shoulder, axilla and flank. Breitner,[18] who studied four cases of traumatic hernia with late manifestations, describes two courses which the trouble may take after recovery from the initial dyspnœa, nausea, inability to eat and general feeling of illness. In the one, after a period of months during which the patient has complained only of vague gastric pains, independent of taking food, and of occasional dyspnœa, symptoms of high intestinal obstruction appear suddenly, following some physical exertion beyond the ordinary. In the other, there are repeated attacks of vomiting and severe pain in the upper abdomen on the side of the injury, and these attacks always come on after ingestion of an unusually large amount of food. In the intervals the patient is wholly free from symptoms. In both cases, the pain is particularly severe under the costal

margin on the injured side. The pathologic basis differs for these two clinical pictures. In the first case, "incarceration" of the stomach and transverse colon in the thorax, with torsion of the stomach to 180 degrees around the sagittal axis and a slight degree of torsion around the vertical axis. In the second case, there is incarceration of a gastric-wall hernia in an opening in the diaphragm.

A cause of traumatic hernia that is, apparently, only beginning to be recognized, but that is of interest to the surgeon from a double point of view, is accidental slitting of he diaphragm during operation of the thorax. Hedblom [4] in a review of 378 cases found three such. In what can scarcely be described as more than a glance through the very extensive recent literature on diaphragmatic hernia, I have come upon three cases in which this etiology is given as probable or possible. Two of these cases were published since Hedblom's review.

MacMillan [19] relates that a soldier had empyema following lobar pneumonia in the left lower lobe. It was drained for two months, after which it healed. Three months after the pneumonia, a röntgenogram was taken to determine the condition of the lung before discharge. It showed the stomach-fluid level and the gas bubble in the left chest. Examination with barium showed an hour-glass stomach with the fundus above the diaphragm and the splenic flexure in the thoracic cavity. The patient's only complaint was slight dyspnœa. There was no history of trauma in this case, the man had not seen action. The hernia was either congenital or due to injury to the diaphragm at operation and MacMillan holds the latter origin to be the more probable.

Adamy [20] reports the case of a woman, aged thirty-seven, who was operated on for post-influenzal pleural empyema on the left side, with resection of the sixth rib. Because of persistent retention of secretion, counterdrainage was established in the fourth interspace anteriorly, above the left breast. A fistula formed and did not close until two and a half months after operation. About one year after the operation, the patient began to have frequent attacks of pain in the left chest, worse on deep inspiration, and radiating into the left side of the neck. There were dyspnœa and palpitation on exertion and weakness and formication in the left arm. She could sleep only on the left side. Physicians consulted laid the symptoms to adhesions and shrinking of the pleura. When the patient was examined by Adamy, these symptoms had continued undiminished for seven years and had lately become worse. Auscultation left the question open between echinococcus of the lung, residual pneumothorax with exudate, and diaphragmatic hernia. The results of percussion of the heart borders proved puzzling. The first examination showed displacement of the heart to the right by one and one-half fingerbreadths, but twelve hours later it was found much farther to the right. The patient said she often had rumbling under the operation wound, but a history of trauma was absent and there had been no severe peritoneal complications at the time of operation. Röntgen examination showed a large part of the stomach and the splenic flexure of the colon above the diaphragm. The lowest part of the stomach filled first; by palpation the contrast mixture could be pressed into the upper part. With the patient lying down, the contrast mixture filled the upper portion completely. The greater curvature was directed upward, the stomach being twisted on its axis about 180 degrees. The difference observed in the position of the heart at the two examinations was now understood as depending on the state of distention of the stomach. The paræsthesias in the left shoulder region are explained as distant phrenic nerve symptoms. This nerve has sensory endings in the peritoneal covering of the diaphragm, which anastomose with nerves supplying the neck and shoulder. Pain in the shoulder has been noted repeatedly in diaphragmatic hernia.

Since the history presents no basis for any other theory of etiology, Adamy believes that injury to the diaphragm at operation must be assumed as the cause of this hernia. He thinks it probable that the injury was confined to the diaphragmatic pleura and the muscular portion of the diaphragm, the peritoneal layer remaining intact and forming a hernial sac. Operation had not been performed at the time of the report. This case, Adamy points out, teaches the danger of exploratory puncture with unclear pulmonary findings.

The third case was published as a case record of the Massachusetts General Hospital, discussed by McAllester, Bock and Jones.[21] A woman had pleurisy at the age of thirty-one. The effusion was drained. She is now fifty and she says that for "over fifteen years" she has had "bilious attacks" occurring about once a month and lasting two days. The attacks usually begin at night, with nausea, vomiting, palpitation, some headache and dizziness. She has dyspnœa and palpitation on exertion and frequently she has pain under the left lower ribs. The bowels are irregular. The hæmoglobin is 60 per cent., erythrocytes 4,100,000, blood smear normal. Röntgen examination shows about half of the stomach passing into the chest through the œsophageal opening. McAllester suggests that the diaphragm may have been injured when the pleuritic effusion was drained.

Bock calls attention to the anæmia in this case and says that he has seen three cases with blood picture of severe secondary anæmia and bleeding from the gastro-intestinal tract in which diaphragmatic hernia was the only pathologic finding. In the literature on diaphragmatic hernia he has seen no mention of gastro-intestinal hæmorrhage. Sisk,[1] it may be pointed out, found occult blood in the stools in one of his cases.

On leaving this discussion of the various ways in which there may come about a diaphragmatic opening that can give rise to hernia, something may be said of that force which drags the abdominal viscera up through the aperture, overcoming gravity and even anatomic anchoring. This force is generated by the difference between the pressure in the thoracic cavity and that in the abdominal cavity. It is the combined influence of pressure and suction, acting with every breath that is drawn, that causes a hernia, once started, to get progressively worse and that creates a hernia out of a weak spot in the diaphragm.

The natural tendency of the organism to set its house in order even in the face of great odds is thus beautifully exemplified in the case reported by Stimson and recently cited by Truesdale,[22] in which a hernia of the entire stomach, demonstrated beyond question in an infant of eleven months, is no longer present in the same child at the age of eight years. Truesdale believes that with hernia of the stomach in infants the tendency is for the opening to get smaller, provided, of course, that the viscus rides freely back and forth through the aperture. Until röntgen examinations are made routinely of every newborn baby, we shall not know how frequent congenital diaphragmatic hernia really is nor shall we be able to form an idea as to whether or not a considerable percentage of such hernias undergo spontaneous cure in early childhood. However this may be, there is, in spite of the action of gravity, scarcely a single known case, as Sauerbruch[16] points out, in which thoracic organs have descended into the abdomen, even with wide open communication between the two cavities.

The symptomatology, referable to the respiratory and circulatory systems on the one hand, to the gastro-intestinal system on the other, varies with the location of the hernia, with the organs herniated and their condition—whether kinked, twisted or constricted—and with the degree of crowding to which the heart and lungs are subjected. Symptoms may be absent and the physical findings practically normal. The symptoms vary with the fullness of the herniated stomach and intestine, and so with the time of day. They vary with the position of the patient; the attacks are frequently mentioned as coming on at night, *i. e.*, with the patient lying down, when gravity acts to enlarge the hernia if it is mobile in the ring, and to distend with fluid a herniated portion of the digestive tube. The partially herniated stomach may alter its position so that the œsophagus that at one time empties above the diaphragm at another time empties below it.[10] Pain that depends on difficulty in the filling or in the emptying of the portion of stomach above the diaphragm will then appear at different times relative to the taking of food. Volvulus of the herniated stomach is fairly frequent; the mechanical conditions are right for it when the stomach wall is fixed to the hernial ring,[2] and when it is present it adds its own symptoms.

The röntgen examination, though beyond question our best, and often our only means of discovering the hernia, cannot always be depended on. If the hernia is intermittent, the picture may be negative. Röntgen examination may fail also if the lumen of the digestive tube is so constricted in the ring that the contrast substance cannot pass from the lower into the upper portion. The circumstances of a diaphragmatic hernia are so unpredictable that it may be extremely difficult to interpret the röntgen picture correctly. Haberer [2] thought he saw an eventrated left diaphragm with the stomach lying in the raised convexity. What he took to be the dome of the diaphragm turned out to be the smooth, convex greater curvature of the stomach, lying upside down above the diaphragm.

It is generally agreed that all diaphragmatic hernias should be operated on, except œsophageal hiatus hernias with short œsophagus, in which repair is impracticable and the symptoms of which may be held in check by dietary management. Also possibly some other small congenital hernias that are practically symptomless, and certain types of hernia in very young children, in which a waiting policy may seem advisable. The presence of a loop of colon in the chest greatly intensifies the desirability of operation, because of the grave hazard of intestinal obstruction.

Diaphragmatic hernia presents especial dangers in pregnancy and labor.

Schwartz [10] reports a case in which a hernia, the result of a revolver bullet wound, gave active symptoms only during three successive pregnancies. The first pregnancy occurred four years after the injury; four and three years separated the pregnancies. The symptoms consisted of vomiting and pain, in crises, which began, in the first two pregnancies, in the fifth month; in the third pregnancy, in the third month. The woman was spontaneously delivered of a living child in each case. In the first pregnancy she was about to be operated on for a supposed beginning peritonitis, when, on the eve of the day set for the operation, the delivery, and with it immediate cessation of symp-

toms, took place. In the next pregnancy she was operated on for supposed appendicitis. In the third pregnancy hepatic or nephritic colic was diagnosed. The intervals between the pregnancies were symptomless except for pain in the left side on effort, but a year after the birth of the third child a persistent cough developed. It was thought that she had pulmonary tuberculosis and a röntgenogram of the chest was made. The diaphragmatic hernia was then discovered. A year and a half later, again several weeks pregnant, she came to Schwartz. Röntgen examination showed an opaque mass, the size of an adult head, in the left thorax, reaching almost to the clavicle. Schwartz operated and found the hernial orifice, the size of a "five-franc piece", about 8 centimetres distant from the costal margin. The diaphragm was very thin and atrophic. The hernia contained about one metre of ileum, the cæcum and the ascending and transverse colons. There were no adhesions except a few of the omentum. The pregnancy continued, but Schwartz was contemplating interrupting it for fear that the repaired diaphragm would not be able to withstand the additional pressure.

Granzow[23] advises sterilization for all women in the childbearing age who have diaphragmatic hernia and who cannot or will not be operated on. In the case that he observed, an unrecognized hernia, dating in all probability about nine years back, when the woman was run over, with resultant fracture of the dorsal spine, gave no serious trouble during pregnancy (loss of appetite and progressive nausea toward the end, no vomiting). She entered the clinic in labor. On the left side of the chest neither heart nor lung sounds could be detected, but gurgling and splashing were heard. Röntgenologic examination confirmed the supposition of diaphragmatic hernia. It showed the stomach entirely filling the left half of the thorax, the heart, trachea, large vessels and œsophagus being pushed to the right. A second röntgen picture, taken four hours later, showed that the displacement of the thoracic organs had progressed; the right border of the heart was almost at the axillary line. The danger from kinking of the large vessels on greater crowding of the mediastinum from further upward pressure of the abdominal viscera, during the period of expulsion, was recognized, and it was decided to deliver the child by forceps at the end of the first stage. But before this time arrived, the mother became cyanosed. With high forceps an asphyxiated but living child was delivered. The placenta followed spontaneously, but the mother was seized suddenly with abdominal pains, vomited, and died under the picture of asphyxiation. At atuopsy an aperture the size of a hand was discovered in the left half of the diaphragm and in the left pleural cavity were found the stomach, greatly dilated, the spleen, the greater part of the omentum, and the splenic flexure and adjoining parts of the transverse and descending colons.

For the approach to a diaphragmatic hernia some operators prefer laparotomy; others, thoracotomy. Others, again, open both thorax and abdomen by one incision. If intestinal obstruction complicates the situation, Truesdale[24] advises doing a cecostomy or appendicostomy and proceeding at a later stage to the repair of the hernia.

*Ultimate Results.*—Hedblom[4] found recurrences reported in about 5 per cent. of cases. In one of Truesdale's[22] cases there were three recurrences in the course of three years in a child five years old at the time of the first operation. He thinks that the condition of the diaphragm on one side of the line of repair, rather than the method of suture, was responsible. Few writers who report cases give late results. Leriche[25] and Lecene[17] report seven and three year cures, respectively, in cases due to war wounds. Leriche's case was a recurrence. The aperture measured 4 or 5 centimetres. He fastened the internal lip of the orifice to the wall, making a horizontal dia-

phragm; in this manner the entire defect was made to disappear. He suggests as other posssible means in difficult cases; suture of the internal diaphragmatic lip to the soft parts of the thorax, cutaneous autoplasty, disinsertion of costal or posterior attachments of the diaphragm, and paralysis of the diaphragm by means of phrenectomy. In Lecene's case the opening in the diaphragm measured 6 x 4 centimetres but had to be enlarged to effect reduction. It was closed with over-and-over linen sutures.

## REPORT OF CASES OBSERVED BY AUTHOR

CASE I.—Colored girl, age twenty-two, received a stab wound in the left chest in 1924. Seen six hours later. General condition good and no signs of shock. A large piece of the great omentum was hanging from the stab wound between the seventh and eighth ribs in the mid-axillary line. It was evident the knife had penetrated the diaphragm and the omentum had herniated through it.

*Operation.*—The abdomen was opened through an upper left rectus incision. Exploration revealed no injury to the abdominal viscera. An opening was found in the centre of the dome of the left diaphragm. It would admit two fingers. The omentum had passed through it. The contaminated omentum hanging from the chest wound was unfit for reduction. The stab wound was enlarged five inches, the ribs spread, the omentum drawn out a short distance and amputated. The clean proximal segment was then drawn back through the diaphragm into the abdominal cavity. The pleural cavity was carefully packed off and a clean wound two and one-half inches long exposed in the diaphragm. Through the thoracic wound the diaphragm was closed by using a few interrupted sutures and a continuous row of chromic catgut. Both the abdominal and thoracic wounds were closed without drainage. Convalescence uninterrupted. The lung soon re-expanded and she left the hospital three weeks post-operative in good condition. There was no infection of the pleura. Patient was seen a few months post-operative and she was all right. It has been impossible to trace her since that time, now five years ago.

CASE II.—Entered the clinic May 1, 1929. Mrs. W. S. H., white, sixty-five years of age, married, mother of four children. Past history unimportant. Normal menopause at forty-five. Successful hemorrhoidectomy ten years ago. No history of trauma.

Her trouble dates back only five years. Chief complaints were epigastric pain immediately after meals, crowding of gas up under the ribs, choking spells, and six attacks of severe epigastric colic radiating up into the chest and between the shoulders. During the height of the pain she thought her heart would stop. Hypodermics of medicine were required for ease. Belching would sometimes benefit. The attacks of pain would last about one hour or until the stomach was empty. Bothered with some pain under the shoulders between spells. She did not spit up her food. Troubled some with qualitative food dyspepsia of the usual gall-bladder type.

A cholecystogram was attempted but the gall-bladder could not be visualized. The clinical history and röntgenologic study of the biliary tract could easily be interpreted in terms of chronic cholecystitis. Ingested barium meal revealed a portion of the stomach above the diaphragm. It was interpreted as a diaphragmatic hernia of the stomach. It was not sufficiently constricted to prevent emptying of the upper loculus. The stomach was fixed (see Fig. 1).

On account of her age, sixty-five years, she was put on medical management and surgery not advised then. That was eight months ago. A letter from her last week indicates that she is getting along quite well and the spells are no worse. She was told that likely the gall-bladder should be removed and the diaphragm explored if her condition grew worse. Up to the present time she is quite content with her lot.

Case III.—First examined December 14, 1924, T. M. B., white, male, age thirty-eight. Married fifteen years. No children. Early history unimportant. Fourteen years ago at the age of twenty-four years, he was injured in a railroad wreck. Extent of trauma not mentioned but he was considerably bruised. He got along very well until nine months ago when he was injured in a second slight "explosion" in some sort of a railroad accident. The injury was not severe. His occupation was a farmer until four years ago, since which time he has been in the transfer business. His chief complaints on entering the clinic were pains all over the body, a sluggish feeling and some gas and distress in the stomach after meals with belching, which gave relief. No severe attacks of colic.

He was a well-developed man; weight 160 pounds. Blood pressure 122/80. Pupils

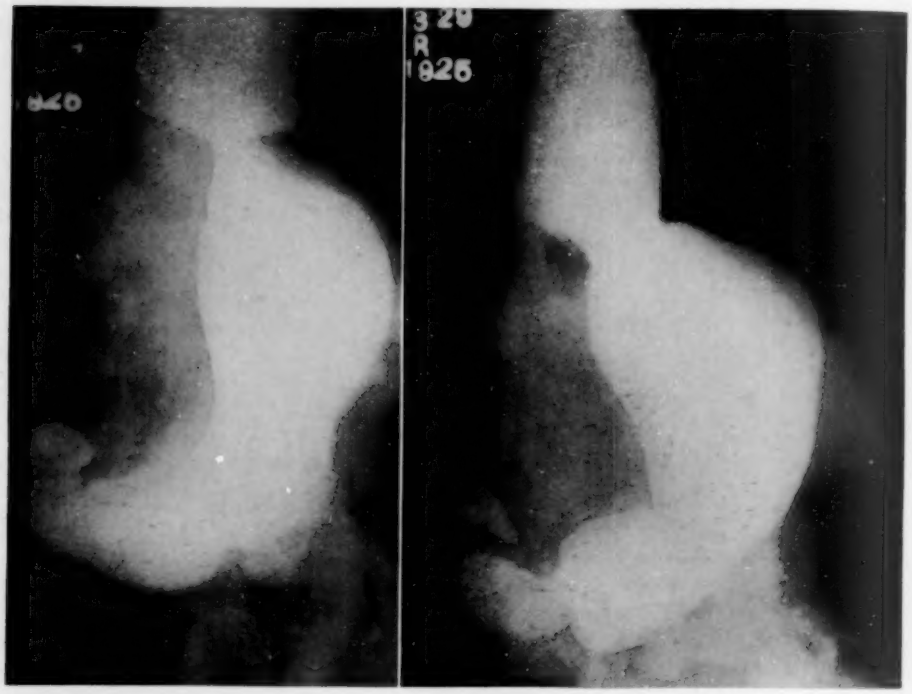

Fig. 1a.—No. 31925. Female, age sixty-five years. Large portion of stomach above the diaphragm. Note the hour-glass effect. Stomach and duodenum otherwise negative.

Fig. 1b.—Same case as shown in Fig. 1a. A few minutes later, when the constriction has lessened and the upper loculus has begun to empty. That portion above the diaphragm is fixed rather firmly.

irregular and fixed. Knee and ankle jerks about normal. Blood Wassermann negative. Spinal fluid four plus in all dilutions. Other findings essentially negative including a complete neurologic examination. Electrocardiographic tracing normal. X-ray of the chest showed the aorta 4 centimetres, right heart 6 centimetres, left heart 6 centimetres. The heart and mediastinum displaced to the right. *Ingested barium meal revealed more than half of the stomach above the diaphragm on the left.* Barium enema revealed the colon within the abdomen.

*Diagnosis.*—(1) Left diaphragmatic hernia with a large portion of the stomach in the thorax; (2) Cerebro-spinal lues.

*Treatment.*—Antileutic remedies have been used since then and he has gotten along very well now for more than five years. Stomach symptoms are unchanged. The position and fixity of the stomach not changed. Surgical treatment was not advised

on account of the associated condition and the lack of severity of his symptoms. Likely trauma played an important part in the etiology of this case (See Fig. 2).

CASE IV.—First examined December 7, 1928, R. F. S., white, female child, five and one-half years of age. The patient was the first child, full term, breast fed, well developed. At 2 and one-half years of age, after a cold lasting a few days, she developed a left empyema. Thoracotomy with resection of two and one-half inches of the seventh rib in the mid-axillary line was done (elsewhere), and a drainage tube was left in several weeks. She was quite sick, emaciated, and coughed a great deal for a number of months. Much pus was raised. About one year after the operation she was seized with a violent attack of abdominal pain with associated nausea and vomiting. No bowel movement for three days. In bed one week and relieved after a large dose of castor

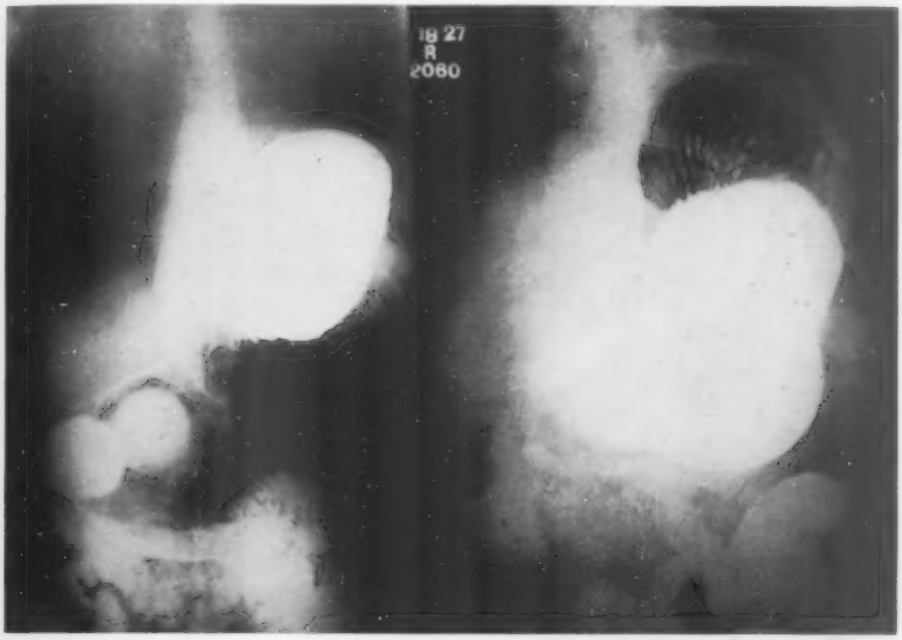


FIG. 2a.—No. 22060. Male, thirty-eight years. Diaphragmatic hernia showing a large portion of the stomach above the diaphragm. Note the incompletely filled lower part. (See also Fig. 2b.)

FIG. 2b.—No. 22060. Male, thirty-eight years. Same case as Fig. 2a. A short time later, when the upper portion of stomach is emptying into the lower and leaving the gas bubble above. About half of the stomach is above the diaphragm and is partially fixed.

oil. Similar attacks have occurred on an average of once a week since. Some spells were worse than others. A hypodermic of some opiate was used for ease in most of the attacks. The spells usually came on late in the afternoon or at night, and lasted several hours. The pain was usually located about the umbilicus, in the episgastrium, and radiating to the left neck. Between attacks her appetite was good and she experienced a feeling of well-being. The spells recurred without reference to diet or bowel movement. She was usually constipated. A great deal of rumbling in the bowels was noticed by the parents when the attack was at its height. The noise could be heard across the room. She preferred to sit or double over during the attack. There was no chest pain nor dyspnœa noted. She took cold easily; head cold now at time of examination.

She was a well-developed and nourished child, height 45 inches, weight 46½ pounds. Hypertrophied tonsils and adenoids, subacutely inflamed. Chronic suppurative maxillary sinusitis, bilateral. Cervical adenopathy. Urine and blood negative. The

lungs on the right side were normal; left, breath sounds clear above the old thoracotomy wound.

*X-ray of the chest.*—Seventh rib deformity still visible on the left; chest symmetrically developed; obliteration of left costo-phrenic angle. A large portion of the stomach lay above the left diaphragm; duodenal cap negative. Barium enema shows a loop of the colon above the diaphragm, the proximal portion distended. Evidently a diaphragmatic hernia, with a portion of the stomach and colon in the thorax.

The maxillary sinuses were drained and the tonsils and adenoids removed March 16, 1929. She was soon free of the upper respiratory foci of infection. On April 9, 1929, the operation was done to repair the diaphragmatic hernia.

*Operation.*—Ethylene anæsthesia was given through a positive pressure machine. An upper left rectus incision four inches long was made through which the diaphragm

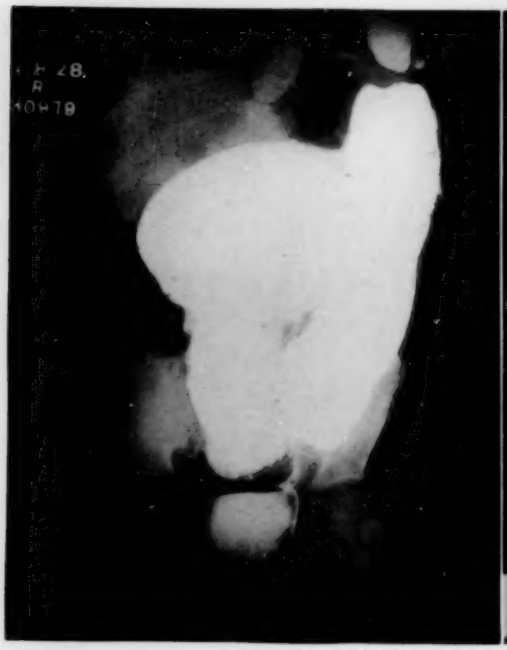

FIG. 3.—No. 30979. Female, age five and one-half years. Barium enema. Loop of transverse colon visualized above diaphragm. Dilatation of proximal segment. Operation with reduction of omentum, colon and stomach into abdomen. Recovery.

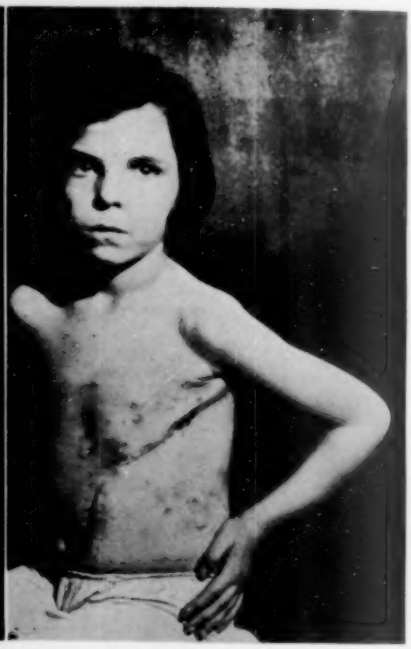

FIG. 4.—No. 30979. Female, age five and one-half years. Same case as shown in Fig. 3. Shows child four weeks post-operative. Uneventful recovery. Note the scars of the combined abdominal and thoracic approach.

was explored. An opening 2½ inches in diameter was found in the left diaphragm a little posterior to the dome. Transverse colon and a portion of the stomach were densely adherent in the opening and could not be withdrawn into the abdomen. An incision was then made over the eighth rib in the mid axillary line extending well forward. A portion of the rib was resected. The former empyema operation had left pleural adhesions which permitted the lung to be pushed well out of the way without opening into the clean pleural cavity. The entire great omentum, a small portion of the fundus of the stomach, and a long loop (8 inches) of the transverse colon nearest the splenic flexure were firmly adherent high in the chest. With one hand in the abdomen, the diaphragm was pushed well into the thoracic wound where the neck of the sac was exposed, and the tissues freed from it all the way around. It was necessary to widen the opening in the diaphragm nearly two inches in order to reduce the hernia into the abdomen. The structures were then easily pulled down by the hand that

was in the abdomen. The edges of the diaphragmatic opening were freshened, and it was closed by using mattress sutures of chromic catgut and a second row of continuous sutures making the approximation smooth and even. All suturing was done through the thoracic wound. There was a false sac of constricting material still about the loop of the transverse colon. It was carefully cut away and the colon straightened out. Both wounds were closed without drainage. The operation was easily and satisfactorily completed and the patient was returned to her bed in good condition. It was a "false" or acquired hernia without a true sac.

The post-operative convalescence was satisfactory. The abdominal wound healed primarily. A small amount of serum accumulated in the thoracic wound but there was no infection. She was entirely well in four weeks. Bowels moved spontaneously and she has never had a cramp since.

May 4, 1929, approximately four weeks post-operative, an X-ray of the chest was made: Left diaphragm high and costo-phrenic angle obliterated; left lung expanded but the lower lobe is still somewhat compressed; no fluid. The entire stomach and colon were below the diaphragm.

She was dismissed from the hospital May 25, 1929, as completely well. A letter from her mother, seven and one-half months post-operative, reports the child in perfect physical condition and still entirely free from cramps. Her bowels move regularly and she is a regular attendant at school. She is now forty-nine inches tall and weighs fifty-three and one-half pounds.

## COMMENT

Diaphragmatic hernia is apparently a more frequent condition than we have previously thought it to be. Many cases are probably never diagnosed because they are not producing symptoms sufficient to urge the person into a complete examination including a röntgenologic study of the gastro-intestinal tract. The combined abdominal and thoracic approach probably expedites the operation and makes it easier and safer for the patient. I have been able to find but few cases recorded in the literature where a previous empyema was the probable cause of a weakened diaphragm eventuating in a hernia. In all probability, my last case here reported is a sequel to such an operation and should be so classified.

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# DIAPHRAGMATIC HERNIA

## Phrenic Nerve Stimulation under Fluoroscope as an Aid in Diagnosis *

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The purpose of this paper concerns itself with the differential diagnosis between diaphragmatic eventration and hernia, with some remarks about the technical procedures in the treatment of the latter condition.

For a long time, diaphragmatic hernia has been a much-discussed abnormality. Curiosity was first aroused by Ambroise Paré [1] in 1610. From then on, occasional cases were observed during the course of post-mortem examinations and were quite fully described by Cooper [2] in his treatise on hernia in 1844. Bowditch [3] assembled eighty-eight case reports in 1883. With the introduction of the röntgenogram, the recognition of diaphragmatic hernia during life has added materially to the knowledge accumulated in regard to the various phases of this subject. Embryologic, anatomic and pathologic considerations have been thoroughly treated by Sailer and Rheim,[4] Keith,[5] and Richards.[6] Analysis of various case reports has been made by Struppler,[7] Griffin,[8] Seibert,[9] Hedblom,[10] and others.

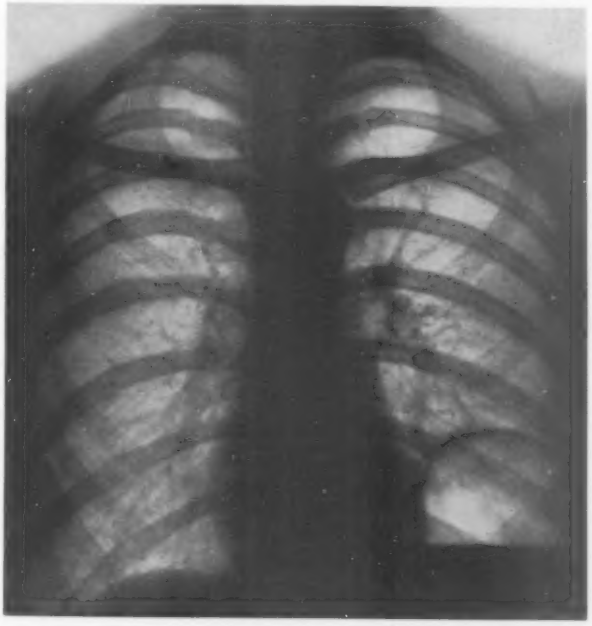

Fig. 1.—Anterior-posterior röntgenogram made before operation. Note thin arched shadow in left lower thorax due to diaphragmatic hernia. During quiet breathing this was stationary and showed only a slight pardoxical movement during forced breathing.

Probably the first case recognized clinically and subsequently operated upon was that reported by Naumann [11] in 1888. A year later, Walker [12] reported a successful operation for a diaphragmatic hernia of traumatic origin. Scudder,[13] in 1912, collected from the literature, fifty-three cases which had been treated surgically. Röntgenologic aspects of this subject have

* Read before the Philadelphia Academy of Surgery, December 2, 1929.

been taken up by LeWald,[14] Uspensky,[15] Carmen and Fineman,[16] and Pancoast and Boles.[17]

All writers have considered diaphragmatic hernia as a condition in which there is a defect in the diaphragm through which abdominal viscera protrude. The defects may be congenital or acquired. They may occur in various parts of the diaphragm, through normal openings, through weak portions due to the lack of development of the musculature or to failure of fusion. If the defect is complete the hernia will not be covered by a sac, and the pleural and abdominal cavities will be in communication unless the opening is sealed by adhesions surrounding the herniating viscus.

Should the protrusion through the diaphragm be a large one, the dome of the hernia may röntgenoscopically give the same appearance as that seen when the diaphragm itself has assumed an abnormally high position. To this condition the term eventration has been most universally applied, although some writers insist that better terms would be diaphragmatic insufficiency, elevation, relaxation "hockstand," etc. The thinning of the diaphragmatic partition is explained on the basis of a degeneration, or congenitally defective musculature, or a paralysis of the phrenic nerve. The difference in pressure relationships between the thoracic and abdominal cavities forces the diaphragm and viscera below it into a higher position within the thorax. In eventration the thin diaphragmatic partition forms, on the röntgenogram, a thin line separating the pulmonary tissue from air collections in the stomach or colon. In hernias the partition is formed by the sac or, if the sac is absent, by the wall of the herniating viscus.

FIG. 2.—Lateral röntgenogram, made before operation, showing large diaphragmatic hernia. Note absence of irregularities in the shadow produced by protruding viscus.

Eventration has been confused with diaphragmatic hernia repeatedly. In fact, Petit,[18] who first described from autopsy findings what was undoubtedly an eventration, applied to it the term hernia, and it was left to Cruveilhier[19] in 1849 to make the distinction. Jaffin and Honeij,[20] in reviewing Bowditch's case, say that he was in error in describing it as a diaphragmatic hernia. Reifenstein,[21] Menville,[22] LeWald, Pancoast and Boles, and other

writers on either subject, emphasize the possibility of confusion and the difficulty in diagnosis. Korns,[23] in concluding a critical analysis of the literature, says, "One finds himself unable to make a definite statement as to the number of genuine cases of eventration which have been studied. The criteria by which the diagnosis has been determined are extremely vague and uncertain in a great many cases."

The importance of differentiating diaphragmatic hernia from eventration is, of course, obvious from the standpoint of treatment. In hernia, gastro-intestinal symptoms of a severe character may be present, and always the potential danger of obstruction is great. Replacement of the herniating viscus and repair of the defect should be attempted. On the other hand,

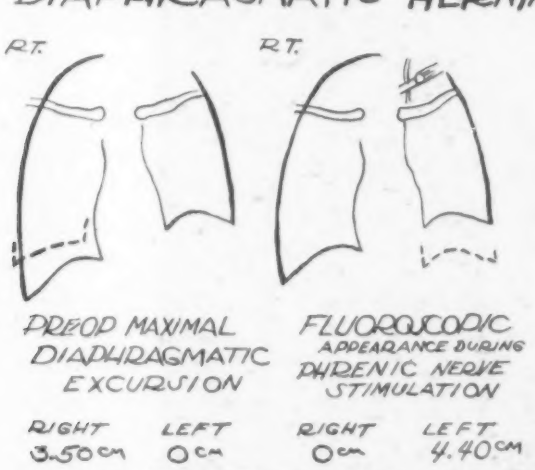


FIG. 3.—Diagrams illustrating diaphragmatic activity before and after left phrenic-nerve stimulation. During forced inspiration the right diaphragm descended 3.5 centimetres, while the left showed no variation in position. Stimulation of the left phrenic nerve caused a 4.4 centimetre contraction.

operation is rarely indicated in eventration, and the propriety of attempting surgical treatment has been doubted by most writers. Lerche,[24] however, planned and carried out an operation for eventration. He plicated the diaphragm with a satisfactory result.

In the differential diagnosis of these conditions a great many of the symptoms and signs are of little value. Etiologically either may be of congenital or acquired origin. The development of symptoms may arise at any time. Pulmonary and gastro-intestinal symptoms are bizarre in both and depend upon the extent of the diaphragmatic disorder and the degree of misplacement of the viscera. As a rule the gastro-intestinal symptoms are less marked in eventration, and intestinal obstruction is rare. Upon physical signs one cannot make a positive diagnosis of either condition. With air in a highly placed stomach or colon so situated because of either a relaxation of the diaphragm or a herniation through it, the high area of tympani and the altered pulmonary signs are confusing in both conditions.

For the exact differential diagnosis between diaphragmatic hernia and eventration the following methods have been applied:

1. *Movements of the Costal Margins.*—Korns has emphasized the value of the observations of Hoover regarding the relation of diaphragmatic activity to the movements of the costal arches. Normally the diaphragm antagonizes the intercostal muscles, so that during inspiration the pull of the diaphragm limits the outward excursion of the costal margin. The higher the diaphragmatic dome or the higher the plane of its activity, the less mechanical advantage it entertains. Korns pointed out that in cases of hernia the costal movements are equal on the two sides because the position of the diaphragm is still unchanged. In eventration the elevated position and faulty antagonism of the diaphragm permit a more noticeable outward excursion of the costal arch on the affected side. It has been pointed out by some that in cases of hernia in which there has been a wide separation of the diaphragmatic musculature, this test may not be reliable.

FIG. 4.—Röntgenogram made after administration of barium meal. Note position of stomach is entirely above cardiac orifice.

2. *Röntgenoscopic Signs.*—In eventration, the diaphragm produces a thin, smoothly curved shadow which is highly placed in the lower thorax. In hernia, this thin, curved shadow (produced by the wall of the viscus or hernial sac) may be irregular in outline. However, if the hernia is large the arching shadow on the röntgenogram may fill the entire hemithorax, and any portion of the diaphragm itself is obscured.

Some observers call attention to the absence of lung-markings behind the elevated diaphragm in cases of eventration. This has been explained by the failure in development of the pulmonary lobes on the affected side. Rheinhold has considered the condition primarily a hypoplasia of the lung, with subsequent faulty development of the diaphragm. This etiological explanation has not been accepted for the majority of the cases reported, and in Lerche's case the bronchi could be seen distinctly beneath the dome of the diaphragm.

Paradoxical diaphragmatic movements may or may not be present in either condition. This also applies to shifts in the mediastinum during the phases of respiration. These mechanisms, compensating for the shifts in the

intrathoracic negative pressure on the affected side, are dependent upon so many factors that Reifenstein, Griffin and others have reported different observations.

The administration of a barium meal, with the patient under fluoroscopic observation, is by far the most important of all of the röntgenologic procedures, according to Pancoast and Boles, and Abbott.[25] Carmen and Fineman point out that in hernia the opaque solution will assume a level higher than the cardiac orifice, while in eventration it will never go above that point. From Stein's[26] reproductions, it can be seen that this test is not reliable. Also it may be impossible to visualize that part of the stomach or colon which has assumed a false situation, regardless of the position of the patient.

Repeating the röntgenographic examination one or more times will often show, in cases of hernia, an altered picture, whereas in repeated examinations of a patient with an eventration the findings will be uniform.

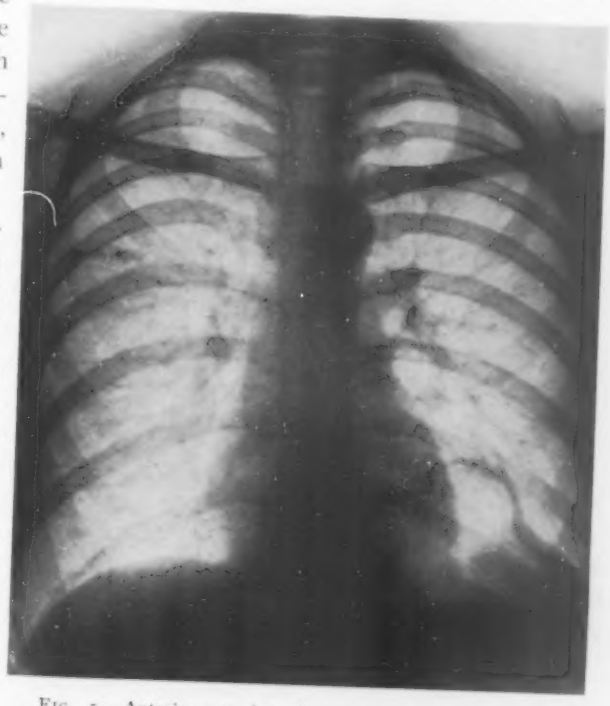

FIG. 5.—Anterior-posterior röntgenogram taken four months after operation for repair of diaphragmatic hernia. The irregular portion of the diaphragmatic shadow is due to an early recurrence of the diaphragmatic hernia. Note elevated position of the diaphragm due to temporary paralysis produced by phrenic nerve crushing. Fluoroscopically the left diaphragm showed no activity.

3. *Intragastric Pressure Studies.*—A method of studying the pressure variations within the stomach, as described by Schliffe,[27] was used successfully by Hildebrand and Hess in establishing a diagnosis in the famous Schneider case. This patient traveled about the clinics of Europe for twelve years before a condition of eventration was diagnosed. In intrathoracic positions of the stomach or in diaphragmatic hernia cases the pressure within that portion of the stomach which is within the chest varies in accordance with pressure variations within the intrapleural space. But if the stomach is still in the abdominal cavity, as in cases of eventration, the findings should be normal, *i.e.*, the intragastric curve rises with the descent of the diaphragm and falls with its ascent.

4. *Pneumoperitoneum.*—This procedure has been used by Verbrycke[28] and others. Lord[29] maintains that it is dangerous, because, if a hernia

without a sac is present, a pneumothorax would result. With our present knowledge of the effects of pneumothorax it would seem that the danger of this procedure has been overestimated.

5. *Laparotomy.*—There are reports of the necessity of resorting to an operation in order definitely to establish a diagnosis. In fact, Lord recommends laparotomy in preference to pneumoperitoneum, a position which hardly seems justifiable.

6. *Faradization of the Phrenic Nerve.*—In the English literature, no record can be found of the use of this method to establish a *positive* diagnosis. Eloesser, in discussing Lord's paper, raised the question as to its practicability. Reifenstein also refers to this method of differentiating diaphragmatic hernia from eventration, but dismisses the subject by saying, "It is difficult to excite in the living individual the phrenic nerve without influencing other viscera." Korns also minimizes the value of such a test by inferring that stimulation of the phrenic nerve cannot be done without influencing other nerves in the neck, scalenus anticus muscle, and other structures. However, Jamin[30] has reported the use of such a method in differentiating eventration from hernia. By exciting the phrenic nerve on the affected side, a response of the diaphragm can be seen under the fluoroscope. In eventration, the Faradization of the nerve fails to cause a contraction. In our case of hernia, the previously immobile diaphragm contracted violently during phrenic-nerve stimulation (see Fig. 3). In Jamin's case of eventration the diaphragm normally had an excursion of five millimetres, and nerve excitation showed no response.

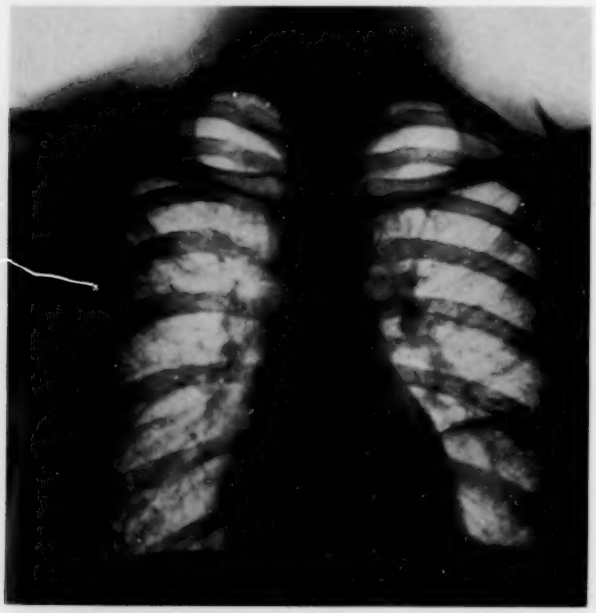

FIG. 6.—Anterior-posterior röntgenogram made eight months after operation for repair of diaphragmatic hernia. Note that the recurrent hernia has greatly increased in size. The effect of the phrenic-nerve crushing had now disappeared, as the hernial sac descended 1 centimetre on deep inspiration, and the faintly visible edges of the separated diaphragmatic muscle moved 2.1 centimetres.

It can be argued that the test would not apply in cases of paralysis of the diaphragm due to a central lesion. This test, however, would never be applied before there was muscle degeneration in the diaphragm, as atrophy would necessarily have to occur before eventration would be present. Andrei,[31] has shown that the atrophic changes following phrenic paralysis

require three to four months and reach their maximum in five months. It has been the observation of Pendergrass,[32] in our röntgenologic department, that in periodic examinations of patients with phrenic-nerve paralysis, eventration is not fully developed until several months after the loss of activity was first noticed.

The study of a patient recently under our observation illustrates well the practicability of phrenic-nerve stimulation, with the patient under the fluoroscope, as a method to differentiate, definitely, between hernia or eventration of the diaphragm.

Case Report.—F. P., aged twenty-two years, female. Admitted to medical service, University Hospital, March 3, 1929. There had been no symptoms until five months previously, when epigastric pain appeared. It radiated to the left and back, was precipitated by the taking of food, and was relieved by the eructation of gas. Recently there had been vomiting of food, poorly mixed with gastric juice. Patient lost twenty

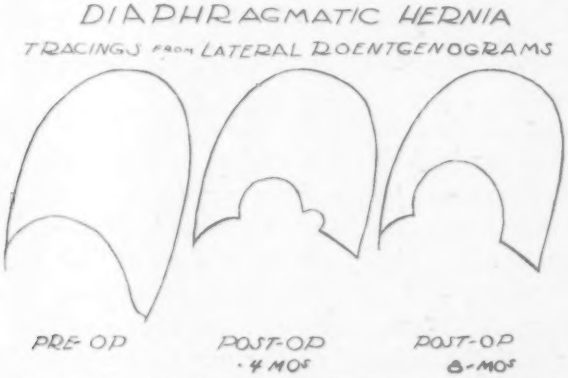


Fig. 7.—Tracings from lateral röntgenograms made before, four months and eight months after diaphragmatic herniorraphy. Note that before operation the herniation was of such magnitude that the diaphragmatic line was obscured. In the anterior-posterior films very little difference between the pre-operative and the post-operative examinations made eight months later can be seen. Notice that in the lateral views the contour of the diaphragm is better visualized.

pounds in weight. There were no other symptoms. The past medical history was negative except for pneumonia followed by a protracted typhoid fever four years previously. The patient had been married two years and given birth to a normal child seven months before admission.

Physical examination was essentially negative except for an area of gastric tympani which extended to the fourth rib in the mid-axillary line and was less marked when the patient was in the erect position. Expansion of the costal arches was equal on the two sides during forced respiration. All laboratory tests, including chemistry and serology, were negative. Fluoroscopically, a thin immobile partition could be seen separating the left chest from the abdomen (see Figs. 1 and 2). During quieter breathing there was no motion on the left dome, and only a slight paradoxic action upon deep breathing. The diagnosis by the röntgenologist was eventration of the diaphragm. After the administration of a barium meal, the entire stomach was found highly placed, with little deformity of its walls (see Fig. 4).

The case was reviewed by another röntgenologist who reported the presence of a large diaphragmatic hernia.

The patient was transferred to the service of Dr. George P. Muller. On March 23 the left phrenic nerve was exposed under local anæsthesia. Under the fluoroscope the

nerve was then stimulated with a minimal threshold Faradic current. The dome descended 4.4 centimetres with each excitation. The nerve was then crushed between hæmostats for a distance of three centimetres. March 26, the abdomen was opened under spinal anæsthesia. A transverse incision in the left upper abdomen was used. Relaxation of the abdominal wall and left diaphragm was well marked. An oval defect was found in the left diaphragm. It was bounded posteriorly by the crura and measured 8 centimetres in length. Through this opening a greater portion of the stomach and great omentum had herniated. These were replaced in the abdomen and the defect closed with two layers of chromic catgut mattress sutures. The patient's convalescence was uneventful throughout. The complete left-sided pneumothorax which was present after operation gradually disappeared, as did also a small pleural effusion.

July 19, 1929, the patient was seen in the follow-up clinic. There were no symptoms. By fluoroscopic examination the diaphragm was elevated and showed no motility. A small recurrent hernia could be noted (see Figs. 5 and 7).

November 15, 1929, a second röntgenoscopic examination was made. The hernia had increased in size and showed 1 centimetre excursion upon deep inspiration, while the edge of the diaphragm beneath the sacculation descended 2.1 centimetres. At this time the patient was still symptom-free and had gained twenty pounds in weight.

*Comment.*—From the study of this case the following points seem to warrant emphasis.

1. Stimulation of the left phrenic nerve under fluoroscopic visualization of the diaphragm provided an exact method to differentiate a doubtful case of a large diaphragmatic hernia from eventration. Röntgenologists had disagreed upon the diagnosis. There appeared in the röntgenoscope a thin, perfectly arched shadow, highly placed on the left side. It was immobile during quiet breathing and showed a slight paradoxical fluctuation during forced breathing. A barium meal was inconclusive, as were the symptoms and physical signs. Stimulation of the nerve supplying the affected diaphragm produced a marked contraction (see Fig. 1). By such a test the integrity of the diaphragmatic musculature was established.

2. Following the tests with the electrode, carried out as an aid in diagnosis, a temporary paralysis of the diaphragm was produced in this case by crushing the phrenic nerve. As soon as it became evident that the case was one of diaphragmatic hernia, this preliminary procedure to the operative repair of the hernia was performed. When the abdomen was opened a few days later the diaphragm was placid, could be easily mobilized for repair, and minimized many of the technical difficulties peculiar to operations upon the diaphragm. Bakes,[33] in 1921, recommended this procedure as a useful form of treatment in patients who could not risk radical operation, as well as a preliminary step to the surgical repair of such conditions. Harrington[34] has also found the method of distinct value.

3. Spinal anæsthesia, in this case, provided good relaxation, an increased tonus of the abdominal viscera, and, hence, more room in the upper abdomen, and a quiet respiration. By the combination of a preliminary diaphragmatic paralysis and spinal anæsthesia, most of the objections to the abdominal approach were therefore overcome.

4. In the first follow-up examination, at the end of four months, a small

recurring hernia could be seen to be forming in the diaphragm that was still functionless because of the preliminary phrenic-nerve crushing. At the end of eight months the activity of the diaphragm was restored, but the hernia had greatly increased in size (see Figs. 5, 6 and 7). This occurrence would seem to indicate that, once a hernia has started to form, the more active the diaphragm, the greater tendency for the herniation to increase in size. The pressure of contracting musculature at the neck of the sac would increase the tendency for impaired function or strangulation of the contained viscus. It is upon this ground that there is a basis for permanent phrenic paralysis in cases of diaphragmatic hernia in whom the operation for the radical repair is contraindicated.

From the experiences in this case it is impossible to say whether permanent paralysis should be induced as a preliminary step in diaphragmatic herniorrhaphy, or if a shorter temporary interruption in function should be carried out by merely freezing the nerve. If the former procedure be carried out, one would convert a case of diaphragmatic hernia, with possibilities of recurrence and intestinal obstruction, to one of eventration. Truesdale,[35] in discussing recurrent diaphragmatic hernia, does not refer to induced preliminary phrenic-nerve paralysis. He concludes, however, that a deficiency in the musculature of the diaphragm exists as a natural sequence of prolonged limitation of function or of trauma to terminal branches of the phrenic nerve in the diaphragm itself. It is quite probable that recurrences are due more to deficiencies in the tissues being sutured than in the suture material itself or in the manner in which they are placed. The technical aspects of the treatment of diaphragmatic hernia have been discussed by Truesdale, Bevan,[36] Soresi,[37] Mayo [38] and others.

5. Emphasis should be placed upon the value of lateral röntgenograms in the study of diaphragmatic lesions. In this case the lateral view shows clearly the sacculation of the hernia, whereas in the anterior posterior view the diaphragm is obscured (see fig. 7). In a recent study of pulmonary ventilation in post-operative patients, Muller, Overholt and Pendergrass [39] found that diaphragmatic studies were only of value when lateral röntgenoscopic or röntgenographic studies were made.

## SUMMARY

Phrenic-nerve stimulation, with fluoroscopic visualization of the diaphragm, aided materially in differentiating a large hernia from possible eventration. Preliminary phrenic-nerve paralysis and spinal anæsthesia simplified the radical operation for repair of the hernia.

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# ACUTE PANCREATITIS

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While acute pancreatitis represents but a small number of the acute abdominal surgical emergencies admitted to the wards of the Mt. Sinai Hospital, New York City, it constitutes a definite proportion of those arising within the confines of the upper abdomen. It is a severe abdominal catastrophe, and one attended with a grave prognosis. It seems strange with the strides which general surgery has made within recent years that the mortality of acute pancreatitis should still be so high. The factors which are instrumental in producing this mortality have been fairly well recognized, but the present method of treatment is far from standardized and it is questionable whether it is either proper or adequate. It is only by a frank clinical discussion of this condition that some light may be shed upon this rather complex, intricate and obscure problem, and with this point in view, a series of fifty-four cases of acute pancreatitis were analyzed. This comprises fifty-one consecutive cases of acute pancreatitis and three secondary to operative interference.

A review of the literature of this condition is unnecessary, for this has been recently covered by Schmieden and Sebening[1] in a very excellent and comprehensive paper.

The etiological factors which cause this disease are still controversial and the methods of infection and portals of entry are open to debate. Innumerable researches have stressed either one or another mechanism, and, when all are considered, it is more than likely that each avenue of infection may be responsible for individual cases.

Theoretically, infection may take place through the ducts, via the common bile duct, the duct of Wirsung, or the duct of Santorini (abetted by direct infection from the duodenum); by way of the lymphatics; through the vascular system by embolism or thrombosis; by contact with suppuration in adjacent viscera; and, lastly, by direct or indirect trauma. Certainly any survey of cases emphasizes the ductal, the vascular and the traumatic etiology. But, pathologically, the œdema, hæmorrhage and pancreatic necrosis present at the time of operation or autopsy often obscure that which may have been the responsible etiological factor in the incipient stage of the disease.

The rôle of pancreatic and bile duct variations as responsible factors has elicited considerable comment and they certainly must play an important part in the production of innumerable cases of pancreatitis. There can be no doubt that in many instances the acute inflammatory changes are due

to those variations which favor pancreatic retention and the retrojection of infected bile into pancreatic tissue. Opie [2] was the first to describe an autopsy of acute pancreatitis in which a small stone lodged in the papilla caused a retrojection of infected bile into the pancreatic duct. This mechanism has been questioned by Mann and Giordano [3] who after a series of careful dissections concluded that the anatomical possibilities of such a mechanism were present in only 3.5 per cent. of their autopsy findings. They concluded that the number of instances is very small in which the anatomic arrangement of the two ducts would permit the passage of bile into the pancreas. Besides, in man the sphincter is located at such a point that when it does contract, both ducts are closed and not converted into a continuous channel. However, in a few cases, a small bundle of muscle fibres is found so located that when contraction takes place both ducts are converted into a continuous channel. While Mann and Giordano admit that experimental investigation has proved an anatomic and physiologic basis for the theory of pancreatic reflux, they believe that the physiologic possibility of infiltrating a pancreas with bile to cause acute pancreatitis must be exceedingly rare. In addition, any mechanism affording the passage of bile into the pancreatic duct will also obstruct the flow of pancreatic juice. Therefore, because of these reasons, the causation for most cases of pancreatitis must be sought elsewhere. Other investigators, however, were able to force bile into the pancreas experimentally by closing the papilla in from 20 per cent. to 66 per cent. of their cases.

No one can deny that gall-bladder disease bears an important relationship to acute pancreatitis. In this series about 85 per cent. of the patients presented pathological evidence of cholecystitis or cholelithiasis. In the fifty-one cases in which the pancreatitis was primary, (not secondary to operative and mechanical trauma) gall-bladder disease was present in forty-one of the forty-two cases in which the gall-bladder was described at the time of operation, and present in three of four autopsies on five unoperated cases. The gall-bladder at operation contained stones in twenty-eight, was enlarged in nine, shrunken and thickened in four, and in only three instances was it definitely described as normal, although two of these cases subsequently discharged stones through a cholecystostomy. On the other hand, in the ten-year period from 1917 to 1926 in which 1280 [4] patients were operated for gall-bladder disease, only thirty-four presented evidence of acute pancreatitis, about 2.66 per cent. This percentage falls well within the conservative 3.5 per cent. of the anatomic possibility of duct arrangement permitting biliary retrojection. In eight autopsies in which the finer anatomy of the ducts was given, six probably presented the variations possible for biliary retrojection. While Schmieden was able to demonstrate calculi at the papilla of Vater in seven of thirty-one cases of acute pancreatitis complicated by stone, in this series a papillary calculus was only encountered in one case although the common duct was dilated in seven instances. But the ampulla of Vater may be occluded either reflexly, or by the œdema and spasm in some cases incident

to the passage of a stone, and in others by inflammatory pancreatic enlargement. No better evidence of papillary spasm or œdema converting the bile and pancreatic ducts into one channel can be found than the occasional case of choledochal drainage attended by pancreatic reflux with the absence of duodenal contents.

Two cases in this series, as demonstrated by autopsy, were undoubtedly due to direct infection from the duodenum or the retrojection of the succus entericus, one via the duct of Wirsung opening separately and directly into the duodenum, and one by the way of Santorini. In the latter instance, the duct of Wirsung was found to be obliterated and the duct of Santorini opened directly into the duodenum.

The lymphatic origin of pancreatitis while defended by some [5, 6] has never been satisfactorily proven experimentally, and certainly the weight of anatomical, experimental [7, 8] and clinical evidence speaks against it. If lymphatic drainage is responsible for cases of acute pancreatitis, why is no instance of it recorded in our two hundred and thirty-five cases of acute gall-bladder disease, and why was it such a rare complication in 1045 cases of chronic cholecystitis and cholelithiasis?

There are a few cases in this series in which the disease occurred in localized areas of the gland. A few of these may have been due to either embolism or thrombosis engrafted on an arteriosclerotic basis.

Operative trauma is not an unusual cause for acute pancreatitis. It has been known to follow operations upon the stomach, duodenum and the gall-bladder. In two cases of subtotal gastrectomy, the injury was due to operative damage to the pancreas in dissecting an adherent perforating duodenal ulcer from the gland in one, and to infection of the tail of the pancreas by a contiguous secondary intraperitoneal abscess. In one case the pancreatitis followed a cholecystectomy with drainage, but the exact mechanism in this case was not discoverable.

The clinical picture of acute pancreatitis was often quite bizarre and the diagnosis was rarely made before operation by most surgeons because the greater frequency of other acute upper abdominal conditions invariably dominated the mind. The fact that most surgeons and clinics have their own individual pathognonomic symptoms and signs by which the condition is suspected proved quite conclusively that the symptomatology is extremely variable. Each case must, therefore, be judged on its own merits. A clinical analysis of the fifty-one primary cases in this series disclosed thirty-three of these sufferers were females and seventeen males. The fourth decade seemed to be the one in which the disease was most prevalent although it occurred in a patient as young as twenty-three and one as old as sixty-nine. The past history of these cases is extremely interesting. A symptomatology of gall-bladder disease was recited by almost half. The typical attacks of right upper abdominal pain occasionally radiating to the shoulder, often accompanied by nausea and vomiting and less frequently by signs of acute infection as manifested by chills and fever were not unusual. Occasionally the history was

that of an ulcer and in fact a few were given medical treatment for this condition.

While 50 per cent. gave a past history of abdominal symptoms varying from weeks to years, the remainder stated their present attack was the first intimation of the disease. The duration of the present complaint varied from a few hours to several days.

The pain in acute pancreatitis was intense and oftentimes so severe as not to be relieved by morphine. In twenty-two patients it was described as originating in the right upper quadrant, radiating to the back in three, and to the umbilicus in one; in eighteen the pain was given as epigastric in nature, often substernal in origin; in nine it was depicted as cramp-like, generalized to the entire abdomen, and in one it was localized to the right lower quadrant, and in one to the left upper quadrant. Vomiting, which was usually severe, was present in thirty-four cases. Twenty-two patients suffered from severe obstipation, and the distention in a large percentage was unrelieved by enemata.

The general appearance of these patients which may be used as an index for surgical procedure seemed to vary directly with the extent and type of pathologic involvement. The average case appeared shocked and acutely ill, and four were in apparent collapse. The pulse though small was not extremely rapid, the temperature was not unduly elevated, in fact a high temperature was unusual, and the respirations were slightly increased. Clinical evidence of jaundice was rare. What the Van den Bergh tests might have shown is not known, but it may have thrown some additional light on the relationship of transient biliary obstruction to the etiology of acute pancreatitis. Cyanosis has been commented upon by others. In this survey it was noted four times. While cyanosis might be due to an anoxemia associated with insufficient aeration secondary to diaphragmatic paresis incident to acute pancreatic pathology, there is still another possible explanation. Inasmuch as the liberated lipase splits fat, there may be an increase in the amount of free blood fat with multiple fat emboli to the lung. As a matter of fact, the frequency of lung involvement in these cases is well known and it is barely possible that the bronchopneumonia occasionally present may be embolic. Three cases in this series were diagnosed on admission as primary pneumonia before it was apparent that the lung involvement was secondary to subdiaphragmatic pathology.

The abdominal examination disclosed tenderness in the right upper quadrant in seventeen, epigastric tenderness in two, localized about the umbilicus in five and generalized throughout the abdomen in eight. Tenderness in the left loin is supposed to be characteristic. This was noted in seven cases. It may be due to distention of the lesser sac with exudate or possibly to involvement of the tail of the pancreas or both. Rigidity was present in the right upper quadrant in thirteen, left upper quadrant in three, confined to the upper abdomen in two, and generalized in eight. Distention was noted in eighteen patients. In seven cases a mass was palpable in the

area of the right upper quadrant, and operation disclosed that this was an enlargement of the gall-bladder in six instances, and in one a pancreatic abscess. Free fluid by physical sign may or may not be present.

The lecucocyte count was high. In 75 per cent. it was over 15,000, and in 36 per cent., above 20,000. The average was about 20,000 white blood cells with a leucocytosis of about 85 per cent. The urine examination was rarely of significance. Bile was present on four occasions and sugar twice.

Abdominal puncture[9] which was employed several times when positive was really of great aid. The intraperitoneal aspiration of a characteristic oily beef juice fluid is almost pathognonomic of acute pancreatitis. It was performed seven times by Doctor Neuhof and Doctor Cohn and this was positive in six instances. This test as an aid in differential diagnosis has not been given the place it deserves in the evaluation of abdominal pathology.

This review of the clinical history and physical examination makes it quite clear that the diagnosis of acute pancreatitis must really be made by exclusion and it must be differentiated from the acute cholecystitis, gastro-duodenal perforations, acute intestinal obstruction, acute appendicitis and other conditions. The text-book differentiations of these are well known and need no discussion here.

When once a diagnosis of acute pancreatitis is made or suspected, the question arises as to procedure. Recently several have advocated quite strongly the non-operative treatment, feeling that the mild cases invariably subside without any sequelæ and that many of the fulminating cases would do better if operated after the disease had localized and abscess formation had taken place and the period of shock had passed. The autopsies of four of five unoperated cases are silent witnesses as to the efficacy of this procedure. There is no doubt that some cases have recovered with the expectant treatment; a few have done well following the drainage of a localized abscess after an acute attack, and several would have died regardless of surgical intervention, especially in those cases in which the entire pancreas seemed almost immediately converted into an œdematous necrotic mass. But what harm is done by surgical exploration? There seems very little to be lost and much to be gained and when it is all weighed, many more cases have probably died from skillful neglect than from active intervention. Acute pancreatitis cannot be considered as a medical disease and the expectant treatment has no more a place here than it would have in acute appendicitis or perforated ulcers.

In this series, the majority of the cases were explored under general inhalation anæsthesia. Spinal anæsthesia, provided that the blood pressure is not too low, is probably the best. Inhalation anæsthesia is to be avoided if possible, especially if any credence is to be placed upon the frequency of embolic lung manifestations. Inasmuch as the majority were diagnosed as gall-bladder disease, an upper right rectus muscle-splitting incision was made, and as a rule this afforded adequate exposure. The findings in the main were quite characteristic. Free fluid varying from a clear amber exudate to the beef broth fluid so characteristic of this condition was present in 45 per

cent. of the cases. It was serous in two, bloody in fourteen, and chocolate colored in four. Its general appearance and consistency may be taken as an index of the stage of pancreatic involvement. Fat necroses which are so pathognonomic of the condition were observed at operation in thirty-two cases and were not noted in fourteen. In one of these, the abdominal wound showed evidences of this after operation, in seven the necroses were intraperitoneal and in seven intra and retroperitoneal. Retroperitoneal fat necroses are a serious complication and their presence increases the degree of protein intoxication. In five autopsies the fat necroses were absent, although three of these were cases of secondary pancreatitis. As is well known, these are caused by the action of the liberated lipase on fatty tissues splitting them into fatty acids and glycerine. The lipase is freed during the digestion of the pancreatic cells by the activated trypsinogen. The free fat then combines with the calcium of the blood and tissue juices to form the characteristic lesion. Their extent, locations, number and size vary from a pin-head to a diameter of several millimetres, but their number and extent is no evidence of the severity of pancreatic destruction. They have been known to suppurate, but in the majority of cases they generally disappear. One case operated years later for chronic cholecystitis showed absolutely no evidence of this condition although at the primary operation the fat necroses were described as extensive.

The gall-bladder, as has been previously mentioned, invariably presents some pathology. The pancreas was described as hard and enlarged in the majority of cases, but after all it is always rather difficult to judge the pathology of this retroperitoneal organ simply by the sense of feel and to visualize it is rather a hazardous procedure in patients sick as these. Occasionally if suppuration was present, the pancreas gave the impression of being elastic or cystic, and in a few cases in which a localized abscess was present, fluctuation was felt.

However, the question of the proper operative procedure is still an open one and a problem which should receive serious consideration. What these patients are suffering from is really an acute protein intoxication, the result of the autolytic action of liberated pancreatic secretion and the toxic products from a necrotizing pancreas. The object of surgical intervention should be the free external drainage of the liberated pancreatic secretions and the products resulting from their digestive action, the possible protection of the pancreas against any further destructions and the removal, if possible, of the factor causing the pancreatitis.

The free drainage of the toxic protein products presents some difficulty. A certain proportion of these poisons may be removed by a thorough and complete suction of the free fluid within the peritoneal cavity at the time of operation. At times, in mild cases, as evidenced by the clinical reaction of the patient and the operative findings, this may be all that is necessary. In severely toxic cases, however, this will not suffice. Ottenberg and Wilensky [10] have suggested exsanguination transfusions to further reduce the protein

intoxication of the blood. But the problem of actual pancreatic drainage is not easily solved. The fact that there are fat necroses means that there must be some free drainage from the pancreas. In some cases, this is sufficient; in others, not. Drainage, however, could be theoretically augmented by a liberal and free incision of the capsule of the pancreas. This is impractical as a rule, not only because of the anatomical location of the gland, but mainly due to the peculiarity of the tissue, which, if traumatized, will digest itself. Pancreatostomy is a heroic procedure and, with the proximity of the splenic vessels, it is fraught with dangers disproportionate to the advantages derived. Three of the four cases in which it was done, died. While incision of the pancreas is not feasible, the peritoneum overlying the pancreas may be bluntly incised and rubber-dam drainage employed. This approach may be either through the gastrocolic or gastrohepatic omentum. This manœuver not only relieves the tension of a swollen œdematous pancreas, but provides an exit externally for the liberated secretions and minimizes the danger of retroperitoneal invasion. This retroperitoneal invasion is a great menace and contributed greatly to cause of death in 33 per cent. of the autopsied cases in which it occurred.

In addition, in these severe cases, a cholecystostomy is usually indicated for more reasons than one. There is no doubt that the surgery of acute pancreatitis has been justly influenced by the association of this condition with gall-bladder disease, and for this reason in any case of acute pancreatitis the gall-bladder and its ducts should receive the most careful and painstaking exploration. In fact many surgeons claim that biliary disease is a precursor of acute pancreatitis, and the prophylactic treatment of acute pancreatitis is the early eradication of preëxisting gall-bladder pathology. As a matter of fact, in this series no case of acute pancreatitis was observed in a patient who had had a previous cholecystectomy. But the treatment of the acute disease should have little to do with the radical treatment of the chronically inflamed gall-bladder. In the majority of primary cases, although it is extremely difficult to prove, the pancreatitis is probably caused by the retrojection of infected bile up the pancreatic duct. This retrojection of bile is probably a transitory condition, for the common bile has never completely occluded as evidenced by the fact that clinical jaundice was present in only four cases even though the common duct was dilated in seven instances. However, it seems safer to afford external drainage of the bile. At the same time, it affords a means of removing stones from a gall-bladder, and in two instances in which the gall-bladder was described as normal and without stones, calculi were subsequently discharged through the cholecystostomy tube—in one case as many as twenty. It is barely possible in these two instances, as in many others, that the origin attack may have been initiated by a stone which was temporarily caught at the papilla and subsequently passed. In another instance in which no stones were found at operation and nothing further done than exploratory coeliotomy, a cholecystectomy was performed one year later for innumerable small stones. In addition, a cholecystostomy relieves tension

and spasm on the papillary region of the duct, thereby promoting a more free drainage from the pancreatic duct, and in those cases in which the common duct is obviously dilated, the external drainage of bile decompresses a compromised liver and protects the pancreas from any further biliary irritation. It seems more than coincidence that the seventeen cases in which cholecystostomy was done for acute pancreatitis should have been productive of the lowest mortality, 35 per cent., while those cases in which exploration with simple drainage was done had a mortality of 55 per cent. While cholecystostomy is rather an indirect way of diverting a stream of bile from the pancreas, the simplicity of its performance has more to commend it than the better drainage obtained by a choledochostomy which is more time-consuming and dangerous. If, however, there is definite evidence of common duct obstruction and dilatation is present, the duct should be thoroughly explored, especially the papillary area, and whether calculi are present or not, choledochostomy is indicated.

Those cases seen late in the course of the disase and in which abscess formation has taken place may be treated as simple intraperitoneal abscesses. Drainage may be performed in either one or two stages. Some surgeons in an effort to avoid soiling the general peritoneal cavity pack down to the abscess and then, after sufficient adhesions have taken place, drain. In those instances in which the abscess has arisen in the tail of the pancreas, a retroperitoneal approach through a lumbar incision has met with success. In this series, four cases of pancreatic abscess were primarily drained, two through the loin, and two abdominally, three of which recovered.

The prognosis in all cases of acute pancreatitis is grave. In this series of forty-six operated cases, twenty-three survived. Seventeen of these made what may be called an uneventful recovery with the exception that one drained bile for rather a considerable period, and the other developed post-operative fat necrosis of the wound. The hospital stay of the average case in this series was about twenty-five days. Six of the cases had rather a stormy convalescence. One case in which a pancreatic abscess was drained through the lumbar route ran a septic course for days, but eventually cleared; another, in addition to signs of a generalized peritonitis, developed a definite left lower lobar pneumonia. Two cases in which the gall-bladder was described as normal at the time of operation subsequently drained stones through the cholecystostomy, and one case was accompanied by the picture of a Charcot fever. One patient sloughed away a great part of the pancreas and it was 110 days before the pancreatic fistula closed. The longest stay was that of over 300 days, in which the pancreatitis followed an automobile accident seven days before admission. Operation disclosed a large hæmatoma of the pancreas which was drained. The patient was acutely ill for months, developing many intraperitoneal abscesses, two of which required subsequent drainage. These abscesses probably followed a peritonitis, although it is barely possible that they resulted from suppurating fat necroses.

Of the cases which died, over half succumbed within the first twenty-four

hours. One case died in five days from an increasing blood sugar and uncontrollable acidosis. Others succumbed on the tenth, twelfth, thirteenth and twentieth days from peritonitis and pancreatic necrosis. One patient died thirty-three days after operation from what was clinically a subphrenic abscess. While the apparent cause of death in 50 per cent. of the cases was shock, autopsy findings in many revealed the picture of an acute hæmorrhage pancreatitis. In fact a careful review of the seventeen autopsies in primary cases demonstrated quite conclusively that death resulted from the actual destruction of the pancreas, sixteen dying from pancreatic necrosis. In six cases the entire pancreas had completely sloughed and was lying in an abscess cavity.

*Summary.*—Acute pancreatitis in this series was coincidentally existent with disease of the gall-bladder as proven by operation or autopsy in 85 per cent. of the cases. However, in a series of 1280 cases of biliary-tract disease exclusive of malignancy or stricture, acute pancreatitis occurred in only 2.6 per cent. of the cases. It seems more than likely, then, that the etiology of acute pancreatitis was dependent in the main upon those variations of the pancreatic and bile ducts which favored anatomically the retrojection of infected bile into the pancreas.

The clinical picture and physical findings of this condition were quite variable, and the diagnosis in most cases must be made by exclusion. When the condition is suspected, a diagnosis might be greatly aided by an abdominal puncture and aspiration of the typical oily beef-juice exudate.

Acute pancreatitis is invariably a surgical disease and operation should aim to remove the toxic protein products, to relieve pancreatic tension and thereby lessen glandular necrosis, and, if possible, to remove the cause of the pancreatitis. Thorough aspiration of the peritoneal cavity, drainage down to the pancreas and cholecystostomy are invaluable procedures to cope with the condition at hand, and in extremely toxic cases, exsanguination transfusions might be of definite value.

Death is due in the majority of cases to the toxæmia of an acute pancreatic necrosis.

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# A CLINICAL STUDY OF CALCIFIED NODES IN THE MESENTERY*

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June 18, 1916, the writer operated on a woman of thirty-one years for recurrent right inguinal hernia that eight years previously had been repaired elsewhere. Nothing first hand was known of what the operator found at that time. For three weeks she had had pain in her right lower quadrant. She thought it due to the hernia because it was worse when the hernia "came out." The pain, however, was not in the region of the hernia, but above it, and was associated with tenderness slightly above and internal to McBurney's point. It was thought that her appendix, descending into the hernial sac, or in some other way, might be the cause of the discomfort. When the internal ring was opened, an atrophic appendix was easily delivered and removed. Before closing the sac, a finger was inserted to explore the pelvis and lower abdomen. A mass like a marble was felt just above the pelvic brim on the right side. A calcified mesenteric node was exposed, measuring 3 centimetres in diameter, to which the omentum was firmly attached. From the node passed a cordlike band to the mesentery of another portion of the intestine. Beneath this band was caught a loop of small intestine, measuring about 35 centimetres in length, moderately distended and very definitely constricted beneath the band.

The omentum was freed, band divided, the loop of intestine released and the node removed. Six months later this woman had gained 16 pounds and was seen nineteen months after operation quite free of symptoms. (Case V.)

This fortuitous finding of thirteen years ago prompted observations on calcified mesenteric lymph glands to see whether they were associated with clinical symptoms that might erroneously be considered something else. This case and several others were included in a paper read at a stated meeting of the New York Academy of Medicine in 1923.

During the past thirteen years, patients complaining of certain types of abdominal pain, and suspected of having calcified mesenteric glands, have had abdominal X-rays taken to discover their presence. In fact, this has become practically a routine procedure of the Presbyterian Hospital and it seems worth while to record the reasons why.

In the first place, the presence of calcified lymph glands in the abdomen, whether in the mesentery or in the retroperitoneal spaces at or near its root, are an indication that at one time or another tubercle bacilli have passed through the intestinal wall and caused tubercles to form, that became scarred, isolated and walled off by calcium salt deposition. Calcification of lymph nodes due to other cause is, by common acceptance, so extraordinarily unlikely that this phase of the subject will not be dealt with here.

How frequently do tubercle bacilli affect the intestine and its glands?

If this be frequent, it is important, for it makes a clinical entity that must be every day taken into consideration.

* Read before the New York Surgical Society, December 11, 1929.

Autopsy figures show that at death intestinal tuberculosis occurs in from 50 to 80 per cent., or even more, of all cases of pulmonary tuberculosis. This means, for example, that of 4780 patients who died from pulmonary tuberculosis in New York City during 1924, 2300 to 3800, or more, had intestinal tuberculosis, a complication seriously affecting their nutrition and, *a priori*, their chance of recovery. In Paris in 1922, 6884 persons died from pulmonary tuberculosis and, of these, 3400 to 5500, or more, had intestinal tuberculosis. Calculated in a similar manner, from 40,000 to 63,000 persons with pulmonary tuberculosis had intestinal tuberculosis at death in the United States in 1923, and from 16,000 to 25,000 in England and Wales in the same year.

Drolet has estimated that about 18,000,000 patients died from pulmonary tuberculosis through the world during forty years. Of this number, from 9,000,000 to 14,000,000 had intestinal tuberculosis.*

These figures, stupendous as they are, refer to manifest intestinal tuberculosis, a disease considered for years as fatal as cancer because diagnosed late, and only, within comparatively recent years, recognized in its earlier stages when more amenable to treatment. Our subject, however, deals with tuberculosis of the intestinal tract that has not killed but been conquered. It is infinitely more common than is generally supposed and begins pre-eminently in childhood.

In Drs. Dunham and Smythe's most excellent paper † in June, 1926, Still's statistics are quoted,

> In discussing abdominal tuberculosis in children, Still ‡ points out that the condition is not common from the clinician's standpoint, "but from the more reliable estimate of the pathologist, abdominal tuberculosis would seem to be one of the commonest of all tuberculous lesions in children." "My own statistics," he writes, "show that 88.3 per cent. of tuberculous children have tuberculous lesions in the abdomen."

and of 120 children with positive tuberculin tests, twenty-one or 17 per cent. showed calcified glands by X-ray, the youngest four years, the oldest 12 and one-half years—average, a little under seven years. More recently Doctor Dunham found that of 1152 children brought for various reasons to the clinics, and routinely X-rayed, 128 showed calcified mesenteric glands, *i.e.*, about 11 per cent.

Abdominal tuberculosis in children should, perhaps, be more common from the clinician's standpoint than it is.

If 11 per cent. of these children showed calcified glands, how many more had tuberculous glands not calcified is hard to say, but it indicates that it must be considered as one of the frequent diseases.

One naturally expects this frequency to greatly vary in different parts of the world. This is borne out by the disparity of figures § between autopsies done in St. Louis, where no evidence of healed tuberculosis was found in 143 cases, children and adults, and 25 per cent. of the British soldiers, who showed these evidences at autopsy.

* Brown and Sampson: Intestinal Tuberculosis, p. 103, Lea and Febiger, 1926.

† Am. J. Dis. Child., vol. xxxi, pp. 815-831, 1926.

‡ Still, G. F.: Common Disorders and Diseases of Childhood, Ed. 3, pp. 423 and 427, Henry Frowde, London, 1920.

§ Opie, A. M.: Rev. Tuberc., vol. iv, p. 641, 1920.

Calcified mesenteric tuberculous glands are plentiful in New York and its vicinity.

There is good reason to believe that calcified mesenteric nodes represent what those most expert in the study of tuberculosis would call Primary Tuberculosis infection as contrasted with the phenomena associated with reinfection or secondary tuberculosis.

Children and young people with tubercule bacilli in their mouths, breathing bacilli into their lungs or swallowing them, have them swept from the tissue spaces beneath their epithelial surfaces through the lymphatic channels to their cervical, tracheo-bronchial, or mesenteric lymph nodes. There they may remain, form tubercles, become walled off and calcified after varying degrees of coagulation necrosis.

Primary infections of this sort in young children may be human or bovine in type. Primary human type infection may occur at any time. Bovine infection seems to be confined to childhood.

PARK and KRUMWIEDE* found in, I abdominal tuberculosis cases, II generalized tuberculosis of alimentary origin, and III generalized tuberculosis, including meninges, of alimentary origin, when the children were under five years, there were 22 instances of the human type as compared with 31 of the bovine. In children from five to sixteen years, there were 10 human and 10 bovine. In adults, sixteen years and over, 21 were human and 4 bovine. Although primary infection to human or bovine is of importance in seeking the source of primary infection, the altered reaction manifested by the body after such a primary infection, or its allergic state, is similar in its response to reinfection in either instance. It would seem, according to those versed in immunity, that reinfection by human tubercle bacilli where the primary infection has been bovine may be quite as disastrous as if it had been human from the start.

ZINSSER† states that "in a community supervised more closely than usual, the work of Public Health Service bacteriologist in Washington revealed 6.72 per cent. of samples of market milk infected with tubercle bacilli. This percentage is probably very much lower than that which would naturally be found in districts with a less well developed dairy supervision, and in some of the poorer farm districts of the country the cattle tuberculosis situation is actually appalling."

These primarily infected nodes may not become so completely walled off, but serve as distributing foci. If bacilli be distributed from them by direct extension, by retrograde lymph spread or through their efferent channels to the blood stream by way of the thoracic ducts, or if new tubercle bacilli be inhaled or swallowed, reinfection of previously existing primary sites may occur. If the immunity provided by the primary infection be adequate, little harm may be done. If the number of new bacilli be great and they gain access to the periphery of already existing lesions, secondary reinfection may cause devastating, often ulcerating and cavity forming, visceral lesions.

The allergic state created by primary infection, *i.e.*, the changed condition of the body and the different manner in which it reacts after an initial infection with tubercule bacilli, together with the amount and virulence of the reinfection, are dominant determining factors in the subsequent clinical

* Jour. Med. Research, October, 1910.

† A Textbook of Bacteriology, p. 506, D. Appleton & Co., N. Y., 1927.

course of tuberculous children as they advance into adolescence. Obviously, it is of importance to recognize the fact that a vast number of children have primary infections, so as to devise every possible way of guarding against serious secondary reinfection. Many cases of calcified mesenteric glands do not show complete walling off with calcium, some have areas of coagulation necrosis and, more rarely, active areas of tubercle formation in their immediate neighborhood. Allergy means altered reaction. Young people with partially calcified glands, may be in an allergic state in relation to tubercle bacilli. The reactions to reinfection they manifest in and about their primary lesion may be and probably are quite different in degree and intensity from reactions shown at this primary infection. That they may show a more intense reaction and perhaps a better immunizing activity if the secondary dosage be very minute, seems possible, but quite the opposite, if the reinfecting dosage be large, so that lesions such as ulcerations, cold abscesses and cavities may result, forerunners of the beginning of the end.

In the gastro-intestinal tract, bacilli little affected by natural gastric juice * and showing "in alkaline media no apparent weakening of virulence," † pass readily by the stomach, where little absorption takes place, and rapidly through the duodenum and jejunum, where food, mixing with digesting fluids, is being prepared for use. The ileum is that part of the digestive tract where the first delay in the contents of the alimentary canal for effective absorption naturally occurs. There, quite as do typhoid bacilli, tubercle bacilli first pass the epithelial barrier to the submucosa and exert their primary action in the solitary lymph follicles or Peyer's patches and make tubercles in the mesenteric nodes. Brown and Sampson depict little mammillations that are the evidences of healed lesser grades of tuberculous infection of the lymphoid structures in the submucosa of the small intestine. Just how many instances of calcified mesenteric glands might show these or less evidence of intestinal wall lesion would require very minute search of the intestinal wall corresponding to the areas drained by the particular nodes involved. The writer at operation has often looked for gross evidences of scarring in the intestinal walls, but found none. This means but little, for the gut was not opened, and even if it had been, the study would have had to be a microscopical one. It is probable that many, if not most, children and young people soon after their mesenteric glands have been infected with tubercle bacilli show no evidence of disease in the intestional wall. One suspects if such mammillations be found that a reinfection of mild degree had occurred in childhood and healed.

Calcified mesenteric nodes are of surgical importance.

1. It is a frequent finding, if a normal appendix be found at an operation for chronic appendicitis.

2. It may closely resemble appendicitis, cholecystitis, gastro-duodenal

* Fernbach, E., and Rullier, G.: Rev. de la tuberculose, vol. iii, p. 160, 1922.

† Baldwin, E. R., Petroff, S. A., and Gardiner, Leroy S.: Tuberculosis, Lea and Febiger, Phila., 1927.

ulcer, intestinal obstruction, renal calculi, diverticulities, pelvic inflammation, vertebral and sacroiliac disease as well as other clinical entities, causing abdominal pain.

3. The need for an operation, the type of incision, and the operative procedure may be determined by a knowledge as to whether they exist or not.

4. They may determine a post-operative result and proper after-treatment.

5. They may indicate an unsuspected advanced abdominal tuberculosis. They indicate that the patient once had a primary tuberculous infection.

6. In an adult they may give the clue to ill health that has existed since childhood.

7. They may be associated with peritoneal bands or adhesions requiring correction.

Calcified mesenteric lymph nodes and other manifestations of abdominal tuberculosis have frequently been experienced by the members of a gathering such as this. Recently Dr. Ross Golden has written an excellent review of the subject.*

Many cases have no clinical symptoms, and the nodes may be discovered when the patient is being X-rayed for something else. Careful analysis of their early history, however, may reveal a very suggestive story of the original infection.

Many cases do present a clinical syndrome. There may be a period in childhood when they were definitely below par, usually under weight, always going to the doctor, always needing a tonic, never quite cured of anything and always convalescing slowly, and peculiarly prostrated with measles or influenza. No diagnosis seems wholly satisfactory—acidosis, grippe, malaria, bilious attacks, intestinal influenza, typhoid fever and appendicitis are some of the mistaken diagnoses used for the early primary stages of these tuberculous infections and their subsequent attacks. They feel their best when living a quiet life in bracing, stimulating climate, during fall of the year and at top weight.

Most cases giving symptoms are seen in young women.

The longer people go with calcified nodes that give no symptoms, the less likelihood there is that they will ever complain. In other words, if symptoms don't appear by young adulthood, they probably will never appear.

Acute abdominal symptoms and signs without leucocytosis or increased polymorphonuclear count should always arouse suspicion of tuberculous mesenteric node infection, whether calcified or not, especially if the pulse and temperature be low.

It is characteristic for these patients, who do give symptoms, to appear never to be wholly well. Just when it would seem that they had swung free, that a period of rest had quite set them on their feet, they crack under the pressure of life's routine. Whatever they ought to be doing, they find they can't quite do.

* American Journal of Roentgenology and Radium Therapy, vol. xxii, no. 4, October, 1929.

To varying degrees, young adults or children with these nodes may be ill-nourished. They may enjoy food, eat naturally, even eat more than their associates, yet remain under weight and lack endurance. They are troubled with abdominal pain. It is not confined to any one place, so that it may suggest many other clinical entities. But it is more often in the right lower quadrant than anywhere else. This is due to the ileocæcal group of glands being involved most often. It may be colic, pain, going as fast as it comes. It may be a persistent, aching, nagging discomfort that sometimes becomes sharp and severe. When this occurs, tenderness accompanies it. This tenderness can usually be accurately charted first by gentle percussion over the whole abdomen as though one were percussing the chest. Subsequently, by pressure with the thumb, a study of deep tenderness is desirable. One of the striking features is to find but little tenderness at McBurney's point. Some may be present there. Inside and above the point, however, is where maximum tenderness is most characteristic. When the pain radiates, as it does not infrequently, it is to the back, "up the back," and into the thigh.

One patient's pain came on after an afternoon of toboganning; another after watching and cheering championship tennis and standing in the train all the way home; another after carrying a heavy weight several blocks on leaving a train at the Grand Central Station; a child played hard and ate apples; one man, seen by an extraordinarily able clinician, was suspected of kidney stone because of the pain in his back and thigh; many young girls after leaving school and its regularity go through an era of strenuous work or exhausting play and then complain. Pain in the past is frequent, and a patient's story cumulates as his memories freshen. Often pain is recognized as an old enemy, sometimes a constant companion. In fact, people may become so used to it as to regard it as natural and fail to appreciate they are suffering. There are many who have attacks, but, when pressed, state they are conscious of "something there" all the time. One young woman in a plaster cast for most of a summer for pain in her back, had a round, smooth shadow in her X-ray film that was entirely overlooked and considered an artefact or a button in her dress.

One of the striking clinical features is the way these patients pay the penalty for physical or dietary stress or strain. While active, during athletic exercise, while keyed up by the excitement of whatever stress may be undertaken, the pain may be absent. Later, when relaxed, the same night, the following day, the penalty is paid. This feature is not invariable, indeed no single symptom is that, but night discomfort after a hard day is too noticeable a feature to be overlooked. These nodes are very close to the great veins of the mesentery, indeed the vessels seem wrapped about them. Venous passive congestion in people with these nodes seems to bring pain when such congestion in people without the nodes does not.

Some cases complain of pain apparently due to peristalsis, so that as soon as they take food they get discomfort that may remain till their next meal.

Many a case has no pain on vacation. Rest, change in work, diversion,

eating different food, breathing different air, change in altitude, and last, but by no means least, opportunity for sun exposure, *i.e.*, those factors that create "metabolic revolutions," greatly influence such cases and are of as suggestive diagnostic, as they are of therapeutic, value.

Vomiting does not often occur, but nausea does, and is usually associated with the attacks of pain. When vomiting does occur, its history may be rather pathetic. Much vomiting in childhood due to unsuspected mesenteric nodes has been wrongly called acidosis. A young man, whose childhood had been shrouded with all the safeguards doctors and protection great wealth could provide, stated there were but few days in childhood that he didn't vomit once in the twenty-four hours. He had come to feel that it was just a bit of his life's routine. In a woman of fifty, precisely the same story was given. Most of her life had been dotted with visits to doctors and sanitoria to find out why she vomited. A loop of ileum was bound fast to her calcified node, so tight that knife dissection was needed to free it.

### HOW THE LESION CAUSES SYMPTOMS

1. Due to adhesions, bands and contractures. These are usually chronic ileus symptoms or symptoms referable to an abnormal tug or strain on the parietal peritoneum wherever the scar tissue may exist. These secondary changes produce symptoms readily appreciated by all.

2. Due to other intestinal and other lymph node lesions of like nature to itself—but active, not scarred nor calcified. The demonstrable calcified areas may be the silent sentinels or indicators of active disease in the neighborhood.

3. Due to the presence of the nodes alone. Apparently this can happen.

It would seem that such lesions indicate a handicapped area in one of the most important functioning areas of the body, and that when the individual as a whole, or the area in particular, is subjected to excessive stress or activity, or infection, it will not function, it breaks down, and so-called intestinal symptoms appear that are not wholly characteristic of any type conditions, *e.g.*, gall-bladder and appendix, we are all familiar with.

1. The lymph and lacteal drainage for the corresponding part of the gut has been diseased beyond repair. The node has been walled off as a foreign body as well as the tissues can do so. A rearrangement and compensatory mechanism has had to be established. The efficiency of such makeshift is generally less than the original arrangement.

2. The same is true of the blood-vessels to and from the nodes and in their immediate neighborhood.

3. Damage may have occurred to the sympathetic trunks in the region and their nutrition been impaired, giving rise to symptoms that may be spastic or paralytic, as the case may be.

4. If there be reinfection, or stress and strain inflicted on such an area, the possibility of rekindling old areas kept in a state of allergy so that intensified, altered reactions of nearly walled-off infection occurs, becomes a

probability. This is, of course, a common happening in the cervical tuberculous glands we can see.

The diagnosis of the presence of these nodes is made by X-ray films of the abdomen, preferably after the large intestine has been made as free of gas and fæces as possible. A good way is to give

| | |
|---|---|
| Castor oil | ½ ounce |
| Elixir lactopeptin aa | ½ ounce |
| Essence of peppermint | 5 drops |

the afternoon of the day before and take the film the following morning. One case showing no calcification at the age of seventeen showed definite calcification at twenty-one. Cases followed in childhood would probably often show this phenomenon. Their flaked, mottled, sometimes ringed appearance, is familiar to all experts in X-ray. It may require repeated films, stereos, transverse as well as antero-posterior views and even filming in prone and supine positions to complete the study. They may be seen by fluoroscopy, but many times when calcification is slight, cannot.

Whereas the diagnosis of their presence is relatively simple, the question as to whether they may be causing symptoms in any individual case is not.

A truly difficult decision arises in cases where the symptoms resemble disorder in the appendix. If the appendix has been removed, this, of course, does not obtain. There are many instances, however, when a patient is referred to the surgeon with a diagnosis of so-called appendicitis and an X-ray film shows calcification to the right of the lower lumbar vertebræ. In most of these cases, true inflammatory attacks with fever, leucocytosis, vomiting, persisting tenderness, and perhaps spasm have been absent. If inflammatory attacks have occurred, the nodes are less under suspicion. Occasionally, if the appendix fills with barium in a routine gastro-intestinal study, its failure to empty, its beaded appearance, the possible evidence of fæcoliths, its abnormal shape or evidence of kinking, adhesion, or dilatation, may contribute evidence that it is diseased. But often one cannot differentiate these two conditions in spite of painstaking effort and conservative treatment over a considerable period of time, till an operation be done.

The writer is not a believer in relinquishing one of the most valuable incisions in surgery, the McBurney incision. It is vastly superior for appendicitis to the right rectus incisions in most cases. If calcified nodes be present, however, and the appendix be destined for removal, a right rectus incision is to be preferred. Examination of the mesentery with node removal may be difficult through a McBurney incision, but easy through the right rectus route.

If an apparently normal appendix be found and removed, the question as to whether the node or nodes should be removed becomes acute. If the operator believes, from the clinical story, that the nodes have been a probable cause of the symptoms, if they can be removed readily, and with minimal risk, if conservative treatment has failed, and if there be no evidence of

widespread tuberculosis, it is wise to remove them. There are many, many cases, who never need to have these nodes removed, but there are some who do. This opinion is based on cases that have given symptoms after appendectomy, cases that have had pain referred to other parts of the abdomen than the right lower quadrant, and cases where operation has been performed at a time the patient was having severe and persistent symptoms and the appendix found to be quite normal.

Where associated adhesions or bands are present, these need, of course, to be dealt with according to the principles covering such conditions.

Great care should be taken in excising these nodes to do no more damage than possible to the structures in the mesentery. Small delicate scissors, fine clamps and thumb forceps, with fine needles and catgut are very helpful. If the nodes are behind the posterior parietal peritoneum, they must be approached from in front, but if they are between the leaves of the mesentery, they can often be approached through both leaves, for the nodes lie on either side of the vessels and it makes the dissection simpler. The incision in the mesentery should be sutured precisely, with a continuous Cushing type of suture so as to leave no raw surfaces.

Most cases we have studied have shown no tubercle bacilli on guinea-pig injection. A few have shown them. Most show no evidences of active tubercles, but a moderate number show cheesy coagulation necrosis. Only a few show active tubercles. The condition being described usually represents the terminal stage of a tuberculous infection, but evidences of earlier stages and of superimposed reinfection are not uncommon.

Conservative treatment should precede surgical removal, unless the appendix demands removal. In such cases one may not be able to wait for this to be undertaken. If these nodes be found in a case with true acute inflammatory disease in the appendix or elsewhere they should not be removed. The risk of introducing an acute streptococcus or colon bacillus or other infection into the retromesenteric tissues should not be taken.

Conservative measures comprise—planning a daily life free from excessive dietary or physical stress and strain—sunlight or artificial heliotherapy, rest, diversion, changes in climate, occupation, or environment, if essential, high caloric food intake—cod liver oil and shielding the patient from secondary pyogenic infections or secondary tubercle bacillus reinfection, as far as possible. If there be much colic, it may be wise to use such sugars and carbohydrates that are readily absorbed in the small intestine and never get a chance to get to the colon and ferment. This means more glucose and less of the other sugars and coarser carbohydrate vegetables. Acute attacks of pain may sometimes be controlled by heat, by an enema, or by a dose of aspirin and pyramidon. The ultra-violet light, two to three times a week, has been one of the most valuable artificial measures employed.

From having pain over a long period of years, often diagnosed incorrectly, some patients get thought habits about themselves that are pitiable—discouragement, self-pity, and fear, combined. They act as canaries let out

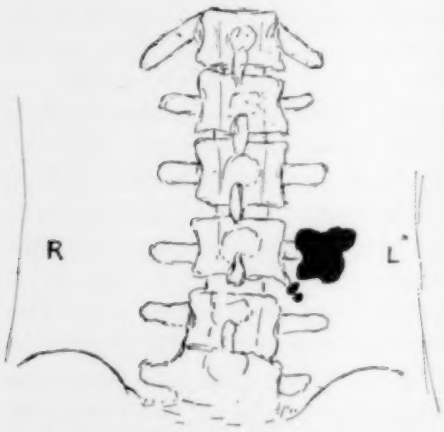
into a room from their happy homes in gilded cages. No matter whether the cage door is open or not they flutter and fly about frightened at what they can't understand.

In such instances wise handling by one skilled in psychiatry may do more good than anything else.

It is not enough for surgeons or physicians to disregard either the significances of calcified mesenteric lymph glands or their presence. They constitute a clinical entity of major importance of frequent occurrence and in carefully selected cases, surgical intervention.

CASE I.—*Calcified nodes resembling acute ileus.*

P. H., No. 39075, trained nurse, a woman, aged twenty-six years, single, admitted September 10, 1918, with history of two main attacks, first one year ago, second six years ago. During the past year lost 30 pounds. One year ago had sudden pain in middle and right side of abdomen for ten days, since when no real attack until six days ago, when in her right side and back, radiating to chest, was a dull, dragging, aching pain, which was made better by drawing legs up, and more noticeable when she stretched them out. She was sent to the hospital from another hospital with a probable diagnosis of acute appendicitis, inasmuch as she had had vomiting on the first night and the second and fourth days following the onset. Her bowels, however, had moved every day. The leucocytes were 6,200, polymorphs 48 per cent. She was considered by the examining physicians on admission as a case of acute appendicitis, gall-bladder disease or possibly twisted ovarian pedicle, but because of the blood-count and inability to localize the tenderness, she was sent to the ward for observation. She vomited bile. On the next day vomiting continued; she had colic, her abdomen was flat, but there was *left-sided tenderness outside umbilicus*, as well as tenderness on her right side. A few red blood cells were found in the catheterized specimen, but subsequent smears and guinea-pig inoculations were negative. Von Pirquet test was moderately positive and was immediately followed by a sudden rise of temperature to 102°, though she had had practically no temperature before, nor did she have any after. Dr. W. W. Palmer saw her in consultation and thought that there was a mild tuberculous process at the left apex. Some infiltration was reported in this region at X-ray. The X-rays of her abdomen showed a large and a smaller shadow in the left abdomen rather characteristic of calcified mesenteric nodes. There was almost no tenderness at McBurney's point.




At operation two masses of calcified glands were removed from the mesentery of the small intestine, near its base. An omental band was divided from the summit of the sigmoid loop. The appendix, which was not inflamed, though angulated at its distal centimetre was removed. A moderate number of other small soft nodes were present in the mesentery. Guinea-pig injection from the calcified nodes was negative.

For over a year she did well and gained 40 pounds and had no further symptoms. Subsequently, in the neighborhood of a year and a half, she was operated on in the Brooklyn Hospital for acute ileus with a resection of eighteen inches of gut, following which she had phlebitis. She twice reported to the Out-patient Department here on account of her phlebitis.

She was last seen 116 months after operation in excellent health.

1. It is hard to explain the right-sided pain except as colic from a partial ileus due to the band to the sigmoid.

2. The tenderness on left side was not noted till late. It was then very definite.

3. The persistence of the vomiting was disturbing and thought to be reflex.

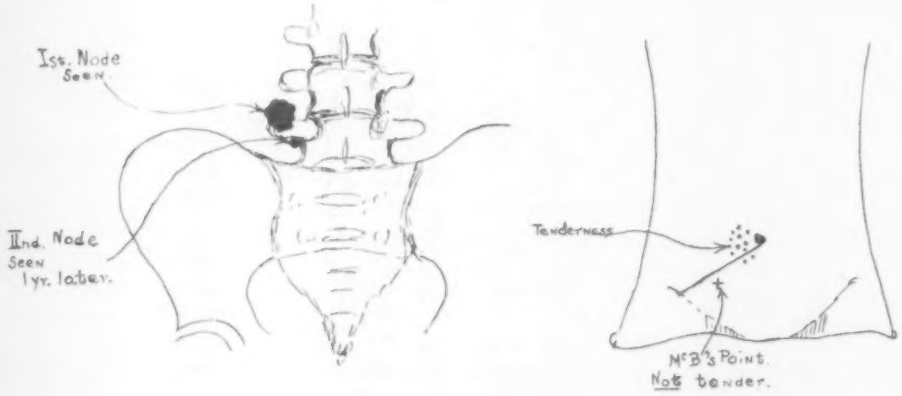
4. The appendix was not inflamed, though slightly angulated.

5. We do not yet know what the pathology of her subsequent ileus was.

She finally died in another hospital of a "streptococcus sore throat."

CASE II.—*Resembling appendicitis—no operation—increasing calcification at nine years.*

P. H., No. 55256, schoolgirl, aged eight years, the daughter of a doctor, besides an occasional stomachache, very slight digestive disturbance and hay fever, had been a well child. Her bowels usually moved every day. On the morning before admission in October, 1922, they had not moved. This day she ate one apple in the morning and three the same afternoon. She was running hard, playing tag, when she began to have colic. That evening she had no alarming focal symptoms and was given a dose of calomel. The next morning she vomited. She had no temperature; her leucocytes were 8,000, but




there was tenderness in the right lower quadrant. She was brought to the Hospital from the country for observation. Her leucocytes on admission were first, 10,400, seventy-five per cent. polymorphs, a few hours later 8,800, seventy-two per cent. polymorphs, then 7,200 and seventy-two per cent. polymorphs on the following day. She was somewhat less well nourished and thinner than the average child of her age. There was definite tenderness above and internal to McBurney's point. She was sent for X-ray on the chance that she might have calcified lymph nodes. A single definite shadow was found to the right of the fifth lumbar vertebra. Her pain and tenderness soon disappeared. She was not operated on. She has been free from further attacks, violent activity having been somewhat lessened and definite attention being paid to increasing her nutrition, cod liver oil, etc. The following year a second X-ray film showed another area of calcification just below the first. The first seemed more thoroughly calcified. She also has calcified glands in the hilus region of both lungs.

N. B.—1. Acute symptoms accompanying dietary and physical stress when bowels had not moved.

2. No fever and no sustained leucocytosis.

3. Tenderness was above and inside McBurney's point, not at it.

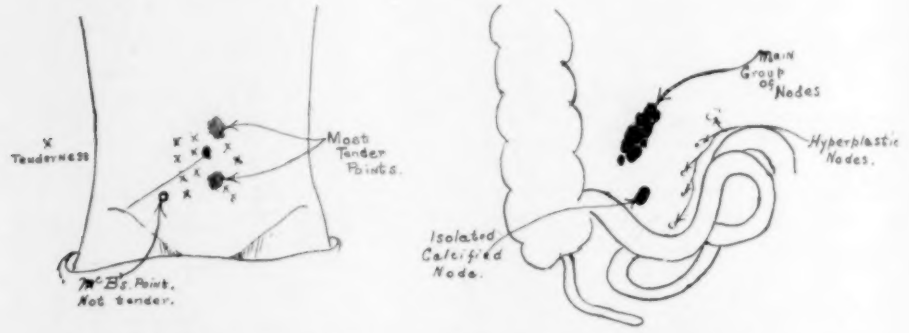
4. No operation was done and for seven years she has not had acute appendicitis.

5. One feels distinctly uneasy about not operating on such cases. They require almost too great discrimination to make it safe not to, and in general it is probably wiser to operate. There were other factors in this case such as her hay fever, a few

sonorous sounds in her lungs, and the fact that she was under her doctor father's close supervision.

CASE III.—*Mistaken appendicitis diagnosis. Calcification occurring between ages of eighteen to twenty-three years.*

P. H., No. 71319, female, single, aged twenty-three, university student. Severe measles with pneumonia at six years. When twenty-one years old severe grippe with marked prostration and swollen neck glands. Tonsillectomy soon after. When twenty-two, began having backache and pain in her right lower quadrant. Pain improved in summer, worse in winter, and was more marked with stress and strain. Abdominal tenderness just above and below umbilicus. In 1923 she was thought to have mesenteric node tuberculosis and sent for X-ray. No calcified areas were found. In 1928 she was considered by two careful and excellent physicians, one a surgeon, to have appendicitis while at college, though she had no increase in her leucocytes nor fever. May 2, 1928, she was X-rayed a second time, four and one-half years after her previous films. Both films, four and one-half years apart, were taken in Dr. Ross Golden's department and read by him. In the later films he found "irregular shadows just above the right ilium and within 3–4 centimetres of the spine, which have the characteristic appearance of calcified nodes."




At operation, May 2, 1928, the appendix was found to be perfectly normal, free, with no constriction, fæcoliths, nor evidences of disease at pathological examination. Above the ileocæcal angle was a group of four or five calcified nodes and another isolated calcified node a few centimetres away. There were hyperplastic glands in the neighborhood. Appendix and glands removed. Active tubercles were not found in the nodes and the injected guinea pig did not show tubercle bacilli.

Noteworthy features: 1. A case where tuberculous mesenteric nodes was diagnosed four years before X-ray films showed calcification.

2. Calcification occurring between the ages of eighteen and twenty-three. Calcification occurs frequently in young children. In this instance it had not occurred at eighteen.

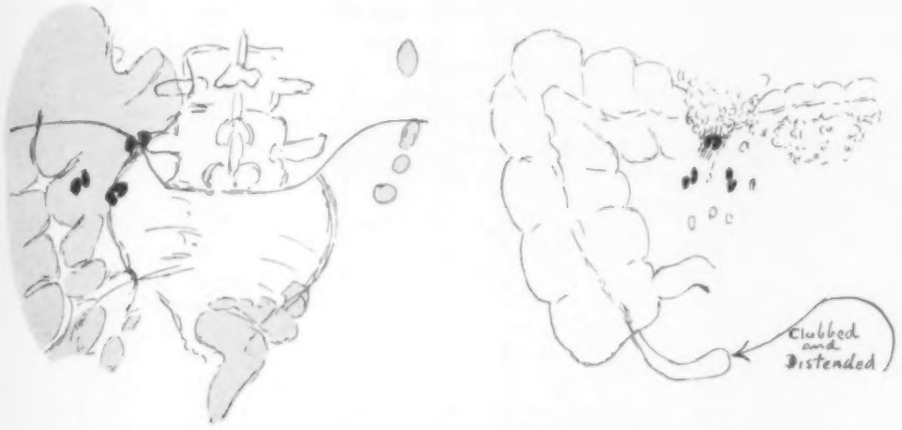
3. The appendix was found indisputably normal in spite of the diagnoses to the contrary before operation.

4. If appendectomy alone had been done in this case, it is more than likely that her symptoms would have persisted.

CASE IV.—*Symptoms due to adhesions to calcified nodes.*

P. H., No. 54290, female, single, aged twenty-two years. Scarlet fever and measles in childhood. Frequent attacks of right lower quadrant pain for a year, radiating to back and pubis, worse with running and other forms of exercise and stretching out at full length in bed, but better with rest, flexing thighs and heat. Leucocytes 5,900, polymorphs forty-nine per cent. On admission June 5, 1922, constipation, appendicitis, tuberculous peritonitis and ureteral callus were diagnoses made. X-ray of her abdomen showed several calcified node shadows. No operation was done at the time, but ten days later she had to return because of the severity of her pain.

At operation the appendix was found clubbed and distended. The first part of the transverse colon was rolled over on its long axis and tightly adherent to one of the groups of calcified nodes causing dilatation of the right colon and appendix. The ileo-cæcal valve was competent. There were too many nodes for removal and the cause of her symptoms was very evident. The appendix was removed and colon freed by sharp dissection and carefully peritonealized. Within six months she gained twenty-eight pounds following intensive rest and nutritional treatment. It is now seven years since operation and she is well, though she still has discomfort with stress or strain.




N.B.—1. A case where adhesions to calcified nodes gave the symptoms.

2. Dilated appendix due to colon obstruction is not rare and may resemble appendicitis, even predispose toward it.

3. Nodes left in. Occasional but no severe symptoms in past seven years.

Case V.—*Ileus from peritoneal band.*

P. H., No. 31243, housewife, married, aged thirty-one years, born in Denmark. Eight years ago operated upon elsewhere for right and left inguinal hernia. Right side

recurred in four years. Three weeks before admission, pain in right side attributed to recurrent hernia. When the hernia was reduced, the pain lessened or disappeared. This pain, however, really did not exist in the inguinal region, but was in the right lower quadrant, where it was associated with tenderness, slightly above and internal to McBurney's point. It was considered possible beforehand that she might be having mild attacks of appendicitis, and that the appendix at times might be descending into the hernial sac. At operation an inguinal incision was made to repair the hernia. The appendix was found to be atrophic, removed, but probably had nothing to do with her

symptoms. Just before ligating the sac, a finger, introduced to explore the pelvis, felt a hard mass like a marble just above the brim on the right side. This mass was pulled into the hernial wound. The omentum was found adherent to a calcified node measuring about 3 centimetres in diameter. From this node a peritoneal band passed across a loop of small intestine to the mesentery of another loop of small intestine, in such a manner as to partially constrict the first loop. There seemed but little doubt but that this was accounting for her symptoms. The band was divided, the omentum freed, the node removed, the hernia repaired, and as soon as she was able to talk after coming out of the anæsthetic, she volunteered the information that the old pain was gone. Six months later, she had gained 16 pounds, her bowels were quite regular. One year and seven months later there were no complaints.

1. A suspicion that the pain was not due to the obvious hernia was substantiated.

2. The pain was probably associated with (1) attachment of the band to the posterior parietal peritoneum, (2) chronic ileus due to incarcerated loop of small gut, rather than appendix, yet there was less pain when the hernia was not down. Whether a descent of small gut aboral to the incarcerated loop might have affected the tension in the latter, is open for conjecture.

3. It was a most fortuitous finding. Undoubtedly, the node would have shown, however, had an X-ray been taken before operation, but it is doubtful whether the band could have been diagnosed before operation.

CASE VI.—*Appendix with fæcoliths. Tubercle bacilli found by guinea-pig infection.*

P. H., No. 70419, girl, single, aged seventeen years, university student. A healthful childhood except that in 1918 during the severe influenza epidemic she was very ill with it. In November, 1927, having once previously had a rather similar attack, she awoke one morning with pain in her right lower quadrant. For over two weeks, this pain kept up, on and off, and she fluctuated between bed in her school infirmary and bed at home. She developed an acute bronchitis. Her highest temperature was 100°. An X-ray showed a calcified node overlying the right margin of the fifth lumbar vertebra.

At operation, December 5, 1927, the appendix was found to contain some fæcoliths, and removed. A partially calcified node, full of cheesy material, was excised from near the base of the mesentery along the ileocæcal vessels. There were no other evidences of tuberculosis elsewhere.

A guinea-pig was injected with material from the node. Acid-fast bacilli were found in the smears taken from its mesenteric gland.

In spite of the fact that the node was proven to be tuberculous, no tubercles nor giant cells could be found in pathological sections made of the patient's node.

The appendix showed no evidence of acute inflammation nor was it tuberculous.

The noteworthy features in this case are:

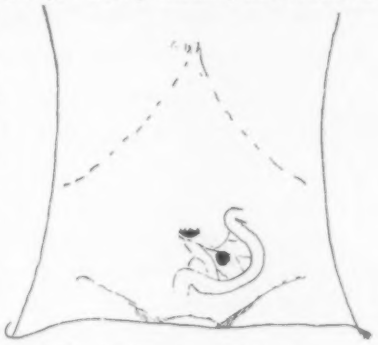
1. The severity of her influenza illness in 1918.
2. Her appendix was a constipated one and probably giving symptoms.
3. The calcified node contained living tubercle bacilli.

CASE VII.—*Left side symptoms.*

P. H., No. 41156, female, single, housework, aged twenty-six years. Perfectly

well up to one week before admission, when she awoke with a nagging, boring pain in the left lower quadrant, with no radiation. This has persisted several hours every day since onset. It was keeping her from work. An X-ray taken by her physician showed a very marked and definite characteristic calcified area to the left of the midline.

At operation, April 12, 1919, the appendix thought practically normal, but removed. The nodes which formed a very definite hard mass were removed. They were in the mesentery just to the left of the midline. Material from them was injected into guinea pig with negative result. She was quite relieved of her pain, and after a somewhat prolonged recovery, due to a hæmatoma in the operation wound, she was discharged in good condition, and remained well when seen several months afterwards.



1. The pain and tenderness, in this case, corresponded to the site of the nodes, on the left side. The pain was of acute character and persisted a week before operation. It entirely disappeared on their removal. There were no adhesions nor other mechanism demonstrable accounting for the pain.

2. The simple presence of the nodes, associated with some exciting mechanism, acting like a hair trigger, was all that could be found to account for her symptoms. The only thing done was to remove the nodes and a normal appendix, yet she was quite relieved.

The above are but a few typical examples of between one and two hundred cases of calcified mesenteric nodes that have been carefully studied during the past thirteen years.

# THE INJECTION TREATMENT OF VARICOSE VEINS*

By Grant P. Pennoyer, M.D.
of New York, N. Y.
FROM THE SURGICAL CLINICS OF ROOSEVELT HOSPITAL

The varicose veins under discussion are those abnormally dilated, superficial veins of the lower extremity. These and conditions resulting from them are a frequent cause of disability in adult life, and any advance in their treatment is important.

The two saphenous systems are the most common to be involved. The great saphenous vein begins on the dorsum of the foot, passes just anterior to the medial malleolus, up the whole length of the medial aspect of the lower extremity to the fossa ovalis. The short saphenous vein passes behind the external malleolus up the posterior aspect of the calf to the popliteal space. These vessels may be multiple and are simply the main trunks of an elaborate network of superficial veins resting in the superficial fascia. It is easier, therefore, to refer to these veins as the greater and lesser saphenous systems. Most of the varices fall into one of these two groups. There are numerous communicating veins between these and the deep veins, especially below the knee. All these vessels are normally supplied with valves composed of single or double cusps, preventing the reverse flow of blood from the hydrostatic pressure in the erect position. Trendelenburg apparently was the first man to emphasize the backward flow of blood in varicose veins. The reflux of venous blood in these large varices without valves is apparently the underlying cause of the deficiency in the nutrition of the involved extremity. If the valves in the communicating veins are also incompetent, as is frequently the case, a vicious circle in the venous circulation is established. Blood escapes from the deep veins through the communicating to the superficial varices. Here, instead of passing upward, it again goes peripherally to be returned to the deep veins to continue the same cycle. It is obvious that this would deprive the superficial tissues of almost all normal circulation and nutrition. Obliteration of the varices breaks the cycle, and immensely improves the circulatory situation. Doctor McPheeters of Minneapolis has recently done some very interesting work on this reverse circulation by observing under the fluoroscope the course of intravenous lipiodol injections. There is no doubt that this is the situation behind many varicose ulcers, and explains the striking benefit often obtained by chemical sclerosis of the neighboring varices. A valuable advantage of this treatment is that one does not hesitate to inject a vein near an ulcer, while excision in such a field would be unwise.

The deeper veins have the support of the muscles. Without some complicating pathology such as phlebitis, back pressure from above, and so forth,

* Read before the Surgical Section of the New York Academy of Medicine, October 4, 1929.

they are rarely involved in the varicose condition of the superficial veins, which have no support. The compression effect of the muscular contractions on the deep veins with competent valves forces the blood towards the heart. This has been demonstrated to be one of the most important factors in the venous circulation of the lower leg, and, of course, is absent in the superficial veins.

The injection treatment of varices seems to date back to the invention of the hypodermic syringe in 1851 by Doctor Pravaz of Lyon. He and rather numerous subsequent workers did considerable work along this line, using strong coagulating and corrosive solutions, such as ferric chloride, iodotannic acid, alcohol and phenol. These solutions which coagulated the blood were early recognized as dangerous, and the treatment was never prevalent or accepted. Sixty years later, in 1911, Professor Linser, of the Tübingen Skin Clinic, noticed the 1 per cent. solution of bichloride of mercury injected in syphilitic patients frequently resulted in firm obliteration of the veins used. He used this solution to sclerose varices with considerable success, and despite its toxic effects, it is still used by a few. Professor Sicard of Paris about the same time used an aqueous solution of sodium salicylate with ideal results, and his work has done more to popularize the treatment than that of any other one man. In 1928 he published a series of 325,000 injections without a single pulmonary infarction. Within the last few years, the chemical obliteration of varices has been taken up by a great many men scattered all over Europe and this country. Its safety and value are now well established.

Numerous solutions have been introduced and which one is ideal is still disputed. Since 1923, 20 per cent. sodium chloride aqueous solution is used in the Tübingen Clinic instead of the original toxic mercuric bichloride solution. Concentrated sugar solutions are effective and popular. Doctor Genevrier in 1921 introduced a now extensively used mixture of solutions of the hydrochloride of quinine and urethane. We have used for almost all our work a 30 per cent. aqueous solution of sodium salicylate, which is made up and sterilized in ampules in the chemical laboratory of the Roosevelt Hospital. Our experience with the other popular solutions has not been sufficient to allow an authoritative discussion of their relative merits. We have been quite satisfied with our results from sodium salicylate. A 5 c.c. injection of 30 per cent. sodium salicylate almost never fails to give a good firm sclerosis of the vein for a very considerable distance even in large varices, and it is our impression that more can be accomplished in fewer injections with this solution than with any other. The obliteration of the vein is rapid, positive, firm and permanent. It has the distinct disadvantage of causing a severe cramp shortly after its injection, which lasts about one minute. Like most of the other solutions, it will cause a slough of tissue if it gets outside the vein in any considerable amount. Quinine and urethane solutions have the advantage of causing no pain following injection. Doctors Kern and Angle of the Johns Hopkins Clinic consider a mixture of equal parts of 50 per cent. glucose and 30 per cent. sodium chloride the ideal solution, because it will

not cause a slough if it gets into the tissues, but we have seen sloughs result from this mixture. None of these solutions coagulate the blood, but depend for their action on an irritative and destructive action on the intima of the veins.

Considerable study has been made of the pathology shown by sections of the veins excised at various intervals following injection treatment. The irritating solution causes a necrosis of the intima. A fibrin deposit, inflammatory cells, and blood clot obliterate the vein lumen in two to four days. A growth of fibroblasts into the fibrin and clot firmly organize it and eventually the vein is converted into a fibrous cord, almost nothing of the original vein structure remaining. The entire process requires several weeks.

Careful comparative studies have been made of the surgical excision and injection treatment of varices. Doctor McPheeters of Minneapolis and Doctor Kilbourne of Los Angeles have recently published extensive statistics, their information being obtained from the literature and questionnaires sent out to many clinics and surgeons. Doctor Kilbourne reported one death from embolism in every 250 cases operated upon, in a series of 4,607 cases. Doctor McPheeters reported almost the same proportion of fatal pulmonary emboli in 6,771 operated cases, and claimed other complications such as pneumonia, brought the operative mortality almost to 1 per cent. Only four authentic cases of pulmonary emboli have been found in injected cases, and almost 400,000 injections have been reported in the literature. Probably all of these four occurred in cases where the injection was done in the presence of a thrombophlebitis, the existence of which is a definite contra-indication to the injection treatment.

The results of the chemical treatment are better. Doctor Kilbourne reports 30 per cent. unsatisfactory results in his reported operated cases and Doctor McPheeters about 20 per cent. Charges have been made that the injection treatment does not give a permanent sclerosis, but this has not been our experience. We have seen only four definite recurrences in our cases followed up to eighteen months. Doctor Kilbourne finds the recurrences in injected cases 6 per cent. New varices occur more often than this, but these are not recurrences of the old varices, and simply mean the underlying cause of the condition is still present. As they occur, it is a very simple matter to inject them, while a second operation is quite a different consideration. The injection treatment is, therefore, safer and gives better results than the surgical excision. No disability is involved in the chemical treatment. The patients continue with their regular work immediately as contrasted to hospital confinement for an average of 15.1 days and an average disability of 34.8 days. No anæsthesia is required, no scars result, the discomfort is less, and the relative cost is smaller.

The details of the technic we have used in our cases are extremely simple, and no special equipment is required. The same needles used to administer salvarsan are employed. They should be sharp and have a rather short bevel. A long bevel on the point predisposes to some leakage of the fluid into the tissues. The ampules of 30 per cent. sodium salicylate solution, a 10 c.c.

glass luer syringe and rubber tubing tourniquets complete the equipment. The site of injection is selected with some care. One must remember that if the leg is dependent, the blood current is reversed, and much of the sclerosis will be below the site of injection. We begin with the lowest varices. Usually at least six to eight inches of obliteration results from each injection of 5 c.c. of solution. If a group of varices involve the entire length of the lower leg, the first injection would be placed about midway between the knee and the ankle. The position of subsequent injections depends upon the results of this first. We have been giving only one injection at one treatment, and waiting to see its full effect before giving the next one. This usually requires almost a week, and it does no harm to wait much longer. Done in this way, rarely more than three or four injections are required to treat completely an extremely involved extremity.

The patient sits on the side of an examining table with the legs hanging over, and a tourniquet is placed loosely above the varices to make them distend further, and the solution is injected into them in the same manner as any intravenous medication is administered. To insure the needle is squarely in the lumen of the vein, it is a good plan to let a little blood flow back into the syringe several times during the injection. The salicylate solution does not coagulate it, and it does no harm to inject the mixture. While and after the needle is withdrawn, firm pressure is maintained at the site of injection to insure against leakage. In a few seconds, a severe cramp occurs, and its location gives some clue as to the diffusion of the sclerosing fluid. With the legs hanging over a table and a tourniquet above the varices, it is usually mostly below the injection point. As soon as the cramp sets in, we remove the tourniquet and allow the patient to lie down on the table. The cramp only lasts about a minute, and no further discomfort is felt by the patient except more or less soreness in the treated vessel until the process is complete. A firm bandage is applied to keep the veins collapsed and should be worn continually until the veins are well obliterated. This is an important point. If the veins are kept quite empty it is frequently difficult later to tell where they were, but if they are allowed to thrombose in the distended condition, they become hard, tortuous, tender swellings which feel like the veins of an infectious thrombophlebitis, and may be troublesome for a considerable period of time. Such a vein may be obliterated from the circulation, but it may be difficult for a time to persuade the patient they have not been made worse by the treatment. Eventually these cases get a good result, but it takes much longer than in those cases where the veins were kept collapsed in the early stages of the process.

The less blood in the vein at the time of injection, the more concentrated and thorough will be the application of the medication to the intima. For this reason, some have attempted to inject them in a collapsed condition. This can be accomplished by having the patient flat, and using tourniquets loosely applied a little above and below the varix under treatment. The needle is inserted with the vein distended. An assistant then evacuates the veins by pressure or elevation of the leg and applies the tourniquets before the injection

is made. Except in very large varices, this is not necessary, and it is much easier to get some leakage of salicylate solution when working with collapsed veins. Occasionally one encounters extremely large varices which are almost like blood sacs, and it is necessary to follow the above technic to get a good result.

The patient, after being bandaged, can resume his or her regular duties without restrictions. They complain of soreness in the treated veins, but no other disability.

We have treated up to date 218 patients, and have given about 500 injections in 306 legs. Forty-eight of these cases we have been able to follow more than a year, and in all these the sclerosis has been so far permanent and satisfactory. All the other cases were injected less than a year ago, or have been lost. Some have returned with new varices. This is hardly an indictment of the treatment, as it means simply that the original predisposing factors to varices are still present, and no one claims the treatment influences these factors. It is a simple matter to inject these new varices, as they are usually only one or two small groups after the main trunks have been removed. I have seen a small number of veins recanalize, nearly all in cases of large varices not well bandaged after treatment, so thrombosis occurred in the distended condition. These were all early cases in the series, and re-injection has given a good result.

Like all treatments, it has limitations and contraindications. It will accomplish easier and better than surgery the removal from the circulation of any superficial vein which we may wish to remove. The end result, as far as the circulation is concerned, is the same as surgical excision, and the procedure is so simple that it can be done with safety in all sorts of cases in which surgery would be undesirable. There are very few factors to be considered except what is to be accomplished by removing the veins in question. Age, general debility, infected ulcers, and so forth, contrasted to surgery, are not contraindications. It is universally agreed it should not be done in the presence of any infectious thrombophlebitis, new or old. The extremely rare reported instances of infarction occur in these cases. Large veins in such cases are frequently compensatory dilatation of the veins instead of true varices. In treating ulcers, it is always to be remembered that many other causes besides the varices are frequently present. Arterial disease, diabetes, syphilis, and so forth, are sometimes factors and they all should be considered before injecting, although none of these conditions are contraindications. Don't expect too much from simple obliteration of varices, if such complications are present. Extremities with chronic elephantiasis and hard, brawny œdema associated with deep extensive ulcers, frequently of years' standing, are little benefited by injection treatment. It is usually hard to find the varices in the thickened œdematous tissue. Sometimes considerable can be done with these cases by putting them to bed for a week to reduce the œdema before injecting, but they will rarely consent to this.

We have not done any work with very small varices. They are not large

enough to be of importance in the circulation. With the use of sheer stockings and short skirts, there is demand for the removal of these small superficial dilated veins for cosmetic reasons, and I do not see why this is not justified. In injecting sodium salicylate solution into veins which are so superficial that they are practically in the skin, there is a tendency for the resulting chemical phlebitis to involve the skin causing small eschared spots which develop into small ulcers which heal rather slowly. Less caustic solutions, such as the sugar and salt ones, are more desirable in this type of case.

We have done very little injecting much above the knee, although many of the men who have had considerable experience, do not hesitate to do so right up to the saphenous opening. Undoubtedly the pressure of extensive varices left above the knee, predisposes to the occurrence of new ones below the knee, but those above the knee do not cause the circulatory disturbances in the leg which are so common in the cases lower down.

Our only complications in the 218 cases have been ulcers, all of which result from faulty technic. I have found only six of these, and four of them were very small and resulted from treating veins which are practically in the skin. Small saccular dilatations in the veins which project almost through the skin, will sometimes give small ulcers even though there is no leakage of salicylate solution. Less necrosing solutions would avoid this difficulty.

The results in ulcer and eczema cases are very encouraging, sometimes dramatic, but not universal. In many so-called varicose ulcers, it is difficult to find the offending varices, and consequently impossible to accomplish much by injecting. When there are large varices present and there is no other complicating pathology behind the condition, the result of obliteration of the varices is sometimes striking. In our series, there are twenty-six cases with long-standing indolent ulcers, who have had prompt healing following the treatment, and have remained healed.

## CONCLUSIONS

Varicose veins of the lower extremity can be permanently obliterated by the injection method.

The method is safer and easier than the surgical excision, and has numerous other advantages making it preferable to the operative treatment.

The technique is simple, and no hospital confinement is required.

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# THE CONTINUOUS INTRAVENOUS ADMINISTRATION OF PHYSIOLOGICAL SALT SOLUTION*

By W. Edward Gallie, M.D. and R. I. Harris, M.D., (By Invitation)
of Toronto, Canada

The administration of physiological salt solution by way of the veins has become one of our most valued methods of treatment. When it is necessary to give it in very large quantities, however, or over a period of several days, the necessity for introducing the needle for each administration constitutes, for children particularly, a very serious difficulty. The method herein described is an attempt to overcome this difficulty.

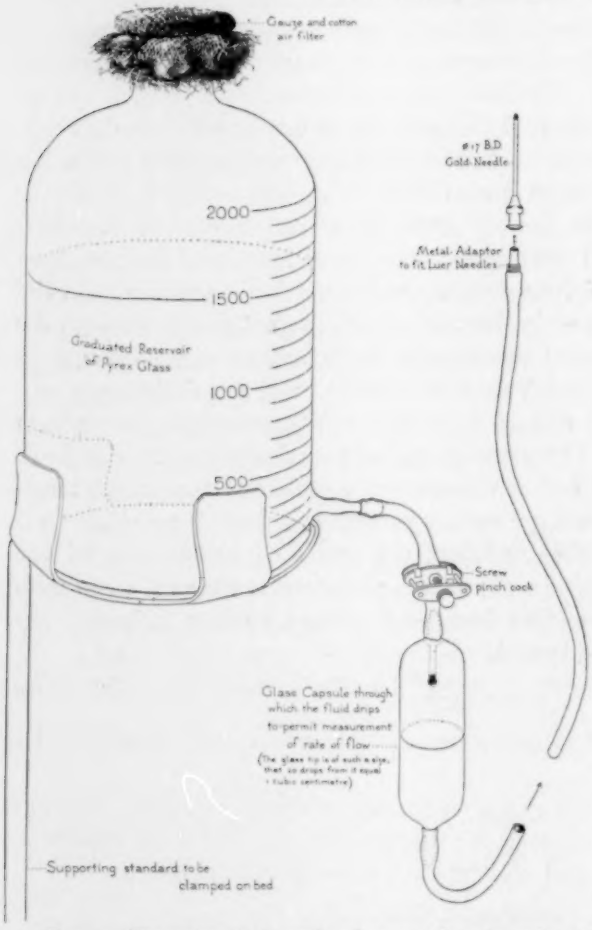


Fig. 1.—The reservoir is made of pyrex glass in order to withstand the temperature of the autoclave in sterilization. The neck is capped with a gauze and cotton pad tied on. This is not removed as long as the flask is in use. When one flask is emptied it is replaced by a freshly sterilized full one. An outlet tube is fused to the side of the flask low down. To this the rubber tubing is attached. The screw cock permits regulation of the rate of flow. The glass capsule through which the fluid drips permits estimation of the rate of flow at any particular moment, since the tip of the cannula in the capsule is of such a size that 20 drops from it equal 1 c.c.

The apparatus is depicted in Fig. 1 with sufficient clearness to render little description necessary. It consists of a graduated reservoir of pyrex glass, fine rubber tubing, a glass capsule, and a fine gold cannula. The pyrex reservoirs are of 2000 c.c. and 4000 c.c. capacity in order to avoid the necessity of frequent changing. It is necessary that they be made of pyrex glass to withstand the heat of sterilization in the autoclave. From an opening low down in the side of the reservoir the fluid passes through rubber tubing to a glass capsule. It drops through this capsule from a glass pipette of such a size that twenty

* Read by invitation before the American Surgical Association, May, 1929.

of the drops falling from it constitute one cubic centimetre. Counting the drops per minute, therefore, renders easy the calculation of the rate of flow at any moment. The rate of flow is regulated by a screw pinch cock. We have found by experience that as small a flow of 500 c.c. per day may be maintained, and of course any rate greater than this.

Before use the apparatus is filled with salt solution and sterilized in the autoclave. The cannula (a No. 17 Becton Dickinson gold needle) is introduced into a superficial vein on the dorsum of the hand or foot (Fig. 2) through a small incision made under local anæsthesia. It is tied in with catgut. If the hand is used, it is well to fix the hand in relation to the

FIG. 2.—This photograph shows the apparatus in use upon a patient suffering from intestinal obstruction following appendicitis. For more accurate delineation of details the dressings have been removed from the hand.

forearm by means of a light anterior splint of plaster or cardboard. This permits the patient to shift the whole arm without endangering the position of the needle. The vein should be small, just large enough to accommodate the needle—since the smaller the vein the faster will be the rate of flow through it for any given rate of administration. This minimizes the danger of clotting. A steady flow should be maintained. Intermittent flow favors clotting in the cannula, while a steady flow fills the vein with saline and prevents clotting.

There are a few points of importance, attention to which will facilitate the use of the apparatus. It is best not to use glucose solutions in this apparatus. This substance, in concentrated solutions, when injected into veins, produces thrombosis with such readiness as to make it one of the most useful of the chemicals used in the injection treatment of varicose veins. Even in the

more dilute solutions (5 per cent. and 10 per cent.), which are so frequently used as intravenous medicaments, prolonged use will also occasionally result in thrombosis. As a routine, therefore, glucose solutions should not be given. If it is considered necessary to administer glucose with this apparatus, the required amount should be given rapidly and should be followed by physiological salt solution. Physiological salt solution or Locke's solution are the most satisfactory fluids for use in this apparatus. (Locke's solution consists of sodium chloride 0.9 per cent., calcium chloride 0.024 per cent., potassium chloride 0.042 per cent. and sodium bicarbonate 0.01 per cent. to 0.03 per cent. It contains all the saline constituents of mammalian blood in their proper proportions.)

No attempt is made to heat the fluid before it enters the vein. To be effective, the heat would require to be applied to the fluid immediately before it entered the vein, since the rate of flow of the fluid is so slow that were heat applied at the reservoir much of it would be dissipated before the fluid entered the vein. To add to the apparatus an appliance for maintaining the fluid at body temperature would add greatly to its complexity. In our opinion, it is simpler to heat the patient by means of hot water bottles than it is to heat the fluid. As a matter of experience, we have found that the administration of fluid at room temperature by this method gives rise to no more disturbance than does the administration of fluid of similar temperature by mouth.

Clotting in the cannula or vein gives surprisingly little trouble if a few simple precautions are taken. As previously mentioned, the vein should be as small as will accommodate the needle in order to ensure as rapid a flow as possible. There should be no cessation in the flow of fluid as this permits blood to regurgitate into the cannula and clot there. It is often useful to permit the fluid to run rapidly for a few minutes to ensure a free channel. If the cannula does become plugged in spite of these precautions, it must be reinserted into another vein.

The use of this apparatus has permitted us to administer large amounts of salt solution over long periods of time. The longest period of continuous use without changing the cannula has been ten days. Continuous administration for two, three or four days has frequently been carried out. The rate of flow may be as low as 500 c.c. per day without clotting.

Discussion.—Dr. J. Shelton Horsley, of Richmond, Va., said that about eight or nine years ago Doctor Matas, of New Orleans, in a paper published in the Annals of Surgery, explained at considerable length a technic that he had been using for the continuous intravenous injection of glucose solution. He used a technic somewhat similar to the so-called Murphy drip in which the rate of dropping could be visualized.

For the last five years the speaker had used Ringer's solution to which 5 per cent. glucose (dextrose) was added. In order to avoid the possibility of air getting into the vein, which of course would be quite a serious com-

plication in the drip method, he was using a long, narrow burette that will hold 500 cubic centimetres of fluid. It is graduated in such a way that the flow can be regulated rather accurately because 100 cubic centimetres covers a graduation distance of 9.2 centimetres. The nurse can tell fairly accurately the rate of flow merely by watching the level of the fluid, and can regulate it with a little screw clamp.

In patients that do not appear to be very sick and where the vein is readily accessible a hypodermic needle can be used, but when the flow is to continue for one or two days we use a cannula and tie it in the vein. In one case he had given as much as 40,000 cubic centimetres. He often gives it for two or three days continuously at the rate of 100 to 200 cubic centimetres per hour. After about two days it begins to irritate the vein, when it is best to take it out, wait for about a day and if necessary insert it again in another vein.

If a solution of glucose or dextrose no stronger than 5 per cent. is injected the vein is not greatly irritated until after it has been used two or three days. The blood normally contains glucose. However, the stronger solutions do tend to irritate the vein.

There are one or two rather important points. The water should be freshly distilled; the tablets for Ringer's solution are added to the dextrose just before it is used. Sometimes he has found a reaction if he has used dextrose that comes in bulk, but there is practically no reaction when the dextrose available in ampoules is employed. By injecting into the tube some insulin at intervals the dextrose is activated and its assimilation is helped. Much nourishment can be given by this method and the normal salts of the blood serum are supplied.

Dr. Alexis Carrel, of New York City, remarked that during the war a smaller apparatus was made for the continuous injection of fluid under the skin or into the vein. This apparatus was constructed along jejunal lines and its great advantage is its extreme simplicity. All one needs is a tube, a needle, and any kind of container for the fluid.

The apparatus consists of a small electric motor which drives a little wheel at the periphery of which there are about six rollers. The tube is placed against a spring and as the little wheel revolves it presses upon the tube. Knowing the number of revolutions, one knows the amount of fluid being injected. It can be regulated to the speed desired.

As nobody was interested in this subject twelve years ago, they manufactured only one apparatus, but it has worked very well. If this method were to be put into general use, it would be very simple to have the little wheel with the motor manufactured in large amounts.

Dr. Dean Lewis, of Baltimore, Md., remarked upon the value of intravenous salt in many lesions. Recently he had a patient with ulcerative colitis. An ileostomy was performed. On several occasions he lost salt rapidly, the blood salt dropping to 286. When the salt was this low the temperature would fall to 96, the patient would become cyanotic and cold.

One of Doctor Horsley's former house officers is on the staff and they gave, as suggested by him, continuous salt intravenously for days. They gave a hypertonic salt solution and Ringer's solution with glucose. The prompt reaction of this patient to intravenous salt given continuously was most striking. The cannula in the vein causes no great discomfort.

PRESIDENT ELIOT asked Doctor Gallie in closing the discussion to touch upon the question of enterostomy, whether in cases in which the treatment was adopted enterostomy was never practised, or whether the treatment was a last resort, and if the treatment failed to secure a renewal of the peristalsis after twenty-four or thirty-six hours enterostomy was then practised. As Doctor McArthur has said, it has proven a life-saving measure in a good many instances, especially where there is not too much delay.

DR. W. EDWARD GALLIE (in closing the discussion) remarked first, for those who have not used the method described in his paper there is one point in the introduction of the cannula which is important. It is wise to choose a small vein, a vein about the same size as the cannula. In that way one avoids having the point of the cannula running around in the vein and injuring it, and it also avoids a backwash.

In fastening the cannula it is rather important, particularly in small children, to devise a method that will allow the patient to move his arm about without taking any risk of dislodging the needle or cannula. He usually made a plaster splint so as to hold the hand in a fixed position, then introduced the cannula into a small vein and then stuck the cannula down with a piece of adhesive plaster. Such an arrangement allows the patient to move the arm about fairly freely and does away with the risk of dislodging the cannula.

Occasionally some patients complain of discomfort in the vein if the fluid is cold. That is not frequent but sometimes does occur. That difficulty can be overcome by putting a hot water bottle on the tube, in the neighborhood of the patient's arm, as it comes to the vein.

Glucose, in his experience, is rather dangerous. That is, it may cause an extensive thrombosis in the vein. It doesn't always do so but it does occasionally.

With regard to his experience with jejunostomy in ileus of children following appendicitis—that is the inflammatory type of ileus—he had used the method a good many times but with no enthusiasm and without any good results. His experience has been that in practically every instance in which he has introduced a tube into the jejunum in this type of ileus nothing ever came out of the tube.

# TRANSACTIONS

OF THE

# PHILADELPHIA ACADEMY OF SURGERY

STATED MEETING HELD NOVEMBER 4, 1929

The President, Dr. Astley P. C. Ashhurst, in the Chair
Calvin M. Smyth, Jr., M.D., Recorder

## BLEEDING ŒSOPHAGEAL VARICES, DUE TO HEPATIC CIRRHOSIS

Dr. Ralph Goldsmith, by invitation, reported the case of a printer, aged fifty-six years, who was admitted to the Jewish Hospital May 4, 1929, in the service of Dr. William H. Teller, to whom the speaker was indebted for the opportunity of operating upon him. The previous history was irrelevant except for the fact that he had been a heavy drinker for about thirty years. December 28, 1928, he had a sudden feeling of weakness and shortly afterward noticed that his stools were tarry. Occasionally thereafter he observed a little blood in his stools, but paid no attention to it. On the day of admission he hurried for a car and suddenly collapsed. He vomited a large quantity of bright blood and continued to do so after admission to the hospital. So profuse was the hæmorrhage that his hæmoglobin fell to 23 per cent., with a corresponding drop in erythrocytes, and his condition was so critical that it was impossible to carry out any gastro-intestinal studies. Physical examination revealed nothing that threw further light upon the case. There was no splenic enlargement. The test of hepatic function showed a normal dye retention. After repeated blood transfusions his hæmoglobin rose to 50 per cent. and operation was performed on May 25, 1929.

The abdominal cavity was entered through an upper right rectus incision. Examination of the stomach and duodenum failed to reveal evidence of any lesion. The stomach was not opened. The liver was definitely cirrhotic, the cirrhosis appearing to be a moderately severe Laennec's process. Additional corroborative evidence was found in that the veins in the gastrohepatic omentum and along the lesser curvature were dilated and unduly tortuous and that the same was true of the veins in the falciform ligament. Accordingly the gastrohepatic omentum was divided between ligatures from as close as possible to the cardia all the way to the pylorus. Its freed edge was then sutured to the anterior parietal peritoneum in the neighborhood of the incision. The operation was done with ease and was well borne by the patient.

Convalescence was entirely uneventful and the patient was discharged in good condition. He has been interrogated at intervals in the interim and has remained well. He was last interviewed on November 1, 1929. At this time he was found to have gained about fifteen pounds. He has been working steadily for several months. He has at no time experienced any discomfort referable to his gastro-intestinal tract, nor has he vomited blood nor passed it by bowel. There was no discernible ascites nor œdema of the legs or feet. He continues to use alcohol, though in moderation as compared with his past indulgences. He considers himself to be in excellent health. It has been impossible to arrange with him to submit to further investigation.

This case was presented with a full realization that the diagnosis of bleeding œsophageal varices was made inferentially rather than by direct evidence, but it is believed that it is as soundly established as is usually the case. It was also realized that the period which has elapsed since operation—five months—is insufficient upon which to base a presumption of permanent success. On the other hand, the rationale of the procedure as well as its apparent safety is such as to warrant the hope that it may be completely successful and to justify the further application of the method.

The reporter remarked that the majority of deaths in patients suffering from portal cirrhosis are undoubtedly caused by hæmorrhage, and the fatal hæmorrhage invariably results from the rupture of varices in the lower portion of the œsophagus. Treatment of this condition has always been unsatisfactory, although a certain number of cases have been benefited by operations of which the Talma-Morrison is the best known and most successful. Rowntree, in the proceedings of the Staff Meetings of the Mayo Clinic, vol. iv, No. 16, April 17, 1929, reported a new procedure in the management of this disease, and presented a case in which it had been utilized. Walters, in discussion, mentioned that it had been carried out in a second case. It is believed that the case herewith presented may be the third to be recorded.

The principle upon which the operation is based is that of preventing the blood dammed back by the portal obstruction from reaching the œsophageal varices. McIndoe, of the Mayo Clinic, injected and removed *en bloc* the portal system and the chief collaterals in some patients who had died of cirrhosis, and found that the bleeding point was practically always situated at the lower end of the œsophagus or just within the cardia. He found, also, that the varices invariably "lie between the coronary vein of the portal circulation on the one hand, and the intercostal and azygos minor veins of the caval system on the other. The coronary vein normally drains the lesser curvature of the stomach from pylorus to cardia, and then turns abruptly to the right within the gastrohepatic omentum to open into the portal vein proximal to its juncture with the splenic vein. Coincidentally with the development of the collateral circulation the flow of blood in this vein is reversed and the full force of the portal current is directed against the cardiac and œsophageal anastomotic venous plexus. A localized area of submucosal varicose veins is produced, which owing to its unprotected situation is particularly exposed to trauma."

The operation proposed by Rowntree on the basis of this mechanism described by McIndow was successfully carried out in two cases by Waltman Walters (*loc. cit.*). It consists of interrupting the flow of blood from the coronary veins to the varices by ligating the former. This is accomplished by ligating and dividing the gastrohepatic omentum throughout its length, care being taken to go as close to the gastric cardia as possible. The divided edge of the gastrohepatic omentum is then sutured to the anterior parietal peritoneum, either incorporating it into the abdominal incision or anchoring it in the region of the falciform ligament of the liver, in the hope of estab-

lishing collateral circulation through the para-umbilical veins to the cava. Thus it is planned to increase the anastomotic routes as well as to prevent further hæmorrhage from the varices.

Dr. Damon B. Pfeiffer asked whether the condition of the spleen was noted at the time of operation. Several years ago in reviewing the literature of gastric hæmorrhage with special reference to the spleen as a cause, he was surprised to observe with what frequency hæmatemesis had been reported by surgeons of the highest qualifications who had nevertheless failed to mention in their reports whether the spleen had been examined at the time of operation or had in any way been considered as a possibility. He reported at that time two cases of massive gastric hæmorrhage due to splenomegaly and based on his observations in those cases made the suggestion, which so far as he can determine was original, that hæmorrhage in these cases is usually due to erosion of the submucous varices in the fundus of the stomach. In that obscure paper he called attention to the fact as stated by Mall that 40 per cent. of the blood in the splenic artery goes to the stomach, and that there is a corresponding venous return. Enlargement of the spleen with its immense demand for blood would probably result in a greater supply to the fundus of the stomach as well, and the well-known capacity of the spleen to contract under certain conditions would from time to time shut into the stomach even greater quantities of blood which would have been carried off by the veins. This would seem to be an efficient cause for the varicosities observed in the fundus. He noted, also, that the vasa brevia were obviously enlarged. He is calling attention to these points in order to induce surgeons, who make reports of operation upon cases of massive gastric hæmorrhage, to note and report also the size and condition of the spleen.

Dr. I. S. Ravdin said that Dr. T. Grier Miller, Dr. Harold Austin, and himself had been engaged in some experimental work on the relation of ligations of the splenic artery and vein to venous pressure in the gastro epiploic veins. They had evidence which indicated that when the splenic artery or vein were ligated at a point distal to where the blood from the stomach emptied into the splenic vein, that pressure rises in the gastro epiploic veins. The speaker suggested that in splenic disease where a condition of this sort might be stimulated within the spleen itself, the rise in pressure in the gastro epiploic veins might produce varices and that this rise in pressure might result in gastric hæmorrhage.

Dr. Hubley R. Owen called attention to a recent report of a case by Dr. Dewitt Stettin, of New York, in which he did remove the spleen. The interesting point of his case was the remarkable return of the blood picture to normal within a very short time. Recently the speaker discharged from the Woman's College Hospital a patrolman who had had two overwhelming gastric hæmorrhages while on duty. The diagnosis was bleeding gastric ulcer. However, it was impossible to operate at that time. He was readmitted to the hospital on September 1. He received practically no treatment

but four or five blood transfusions, after which paracentesis removed considerable fluid from his abdomen. Doctor Owen asked Doctor Goldsmith the blood picture of his patient when he operated. In the speaker's case the hæmoglobin was 40 per cent. with about 2,000,000 red cells.

DOCTOR GOLDSMITH reported that his man was thoroughly explored and his spleen was substantially normal in size, perhaps slightly enlarged. The veins along the lesser curvature of the stomach were enlarged. His liver did not show very marked change; none the less he had these violent hæmorrhages. The speaker had thought of having an examination with the œsophagoscope at some later date, but has been unable to arrange it. The blood picture was 30 or 35 per cent. hæmoglobin which was raised by transfusion until it was in the neighborhood of 50 per cent. The operation itself is extremely simple and was followed by little or no reaction.

## SUBACUTE HÆMORRHAGIC PANCREATITIS

DR. RALPH GOLDSMITH reported the case of a man, aged forty-five years, who had always been in what he considered to be good health. For two years, however, he had been troubled at times by attacks of indigestion which took the form of a feeling of abdominal distention and discomfort. This he attributed to constipation and was able to obtain relief by enemata and carminatives. His appetite was always excellent and he was not particularly distressed by the taking of food. There was no loss of weight or strength. He was very nervous, and when agitated for any reason whatever was in the habit of vomiting. This vomiting sometimes occurred coincidentally with the attacks of indigestion, but was just as likely to happen independently of the latter.

In February, 1929, the patient, in order to ward off a cold, purged himself thoroughly. He was awakened that night by severe cramp-like abdominal pain accompanied by vomiting. He was treated by his physician and the pain wore off gradually, but he felt weak enough to remain at home for two or three days. X-ray examination on March 1, 1929, was reported as follows:

> "Röntgenographic study of the abdomen made six and twenty-four hours following the inspection of an opaque meal reveals evidence of an abnormal spastic type of colon. Fluroscopic examination shows evidence of a localized tenderness over the shadow of the contrast filled appendix. Cholecystography shows evidence of chronic gall-bladder disease. There is still retention of the contrast meal in the caput coli and appendix at forty-eight hours following the ingestion of the meal and still a well defined tenderness immediately over this area. There is undoubtedly röntgen evidence of chronic pathological changes about the appendix."

March 7, 1929, the patient was again awakened by an attack similar to the one described above. The pain was described as "crampy, knife-like and as though a belt were being pulled tightly around the abdomen". It was rather more intense than in the first attack, and was most marked in the right lower quadrant. It was again accompanied by vomiting and sweating, but no fever nor chills were noted. A hypodermic of morphine was administered with considerable relief. A diagnosis of appendicitis was made and on March 17, 1929, the patient was admitted to the Jewish Hospital.

The patient walked into the hospital. He made no complaints and appeared to be entirely comfortable. His temperature, pulse and respirations were normal. Blood pressure was 124/84. Physical examination revealed

diseased tonsils, a lipoma of the shoulder and a very obese abdomen with moderate tenderness of the right side, particularly in the lower quadrant. There was no rigidity, no masses were felt, peristalsis was active, and the matter of shifting dullness was not noted. Examination was otherwise negative. An electrocardiogram showed a simple tachycardia only. The urine was negative except for a trace of albumen. Examination of the blood revealed hæmoglobin 92 per cent., neutrophiles 75 per cent., erythrocytes 4,650,000; lymphocytes 25 per cent., leucocytes 9100. The Wassermann reaction was negative. Blood sugar was .091, blood urea nitrogen 13 milligrams. A diagnosis of chronic appendicitis and chronic cholecystitis was made and the patient operated upon on March 19, 1929. Upon opening the peritoneum there was a gush of creamy pinkish-white fluid containing recognizable globules of fat. So much of this escaped that it was impossible to measure it, but there must have been several pints. There was necrosis of the omental fat and of the appendices epiploica. The peritoneum was reddened and exhibited white fatty plaques. Exploration revealed a rather small, free appendix, not acutely inflamed. The gall-bladder was thickened but contained no stones. The pancreas was enlarged and hard, with areas of boggy softening and necrotic spots. The patient took the anæsthetic very badly and at all times during the operation his condition was a cause of grave concern. For this reason, and on account of the fact that he had been practically asymptomatic prior to operation, it was considered inadvisable to subject him to any prolonged surgical procedure. Therefore drainage was instituted through the original incision and through a stab wound in the left lower quadrant and the abdomen closed. His convalescence was uneventful.

The patient was examined October 30, 1929. His wound was firmly healed, and there was no hernia. He had been working steadily since shortly after his discharge from the hospital and had suffered no inconvenience. His digestion was rather better than before operation and he had had no further attacks of pain. He considered himself well.

Doctor Goldsmith remarked that it seems reasonable to suppose that this man had suffered an attack of acute hæmorrhagic pancreatitis on two occasions prior to operation. The etiology was probably a chronic bile-tract inflammation with extension along the pancreatic duct. It is likewise probable that he would have recovered without operation, since the acute symptoms had subsided before he came to the hospital. It is recognized that because of the non-removal of a diseased gall-bladder he may have a recurrent attack of pancreatitis, but it was felt that a cholecystectomy would have jeopardized his life at the time of operation. He has since refused to have this procedure carried out.

The clinical picture of hæmorrhagic pancreatitis is ordinarily so striking and presents features of such gravity that it is properly considered to be one of the most dangerous of intra-abdominal lesions. That this is not always the fact, however, is indicated by the subjoined case, which is interesting for the reason of the disparity between the clinical symptoms and the pathological findings.

This case is presented to indicate the difficulty in making a diagnosis of pancreatitis on the evidence obtainable, as well as to call attention to the fact that the peritoneum has higher powers of resistance to the pancreatic enzymes than is generally realized. It is likely that many cases of pancreatitis of this character recover spontaneously and are never recognized.

## RECURRENT TUBERCULOUS LYMPHADENITIS OF THE AXILLA AND NECK

Dr. Damon B. Pfeiffer presented a man aged thirty-one years, who first noticed several small enlarged nodes in the axilla and the neck on the right side in the Fall of 1923. They were not painful or sore to touch. On account of their gradual enlargement several months later one of them was removed for examination. The microscopical diagnosis was tuberculosis.

The patient was exceptionally robust and healthy. The family history was negative, and there had been no known exposure to tuberculosis. He had had no illnesses except measles, chicken pox and possibly diphtheria in childhood. Examination of the lungs was negative and has remained so up until the present. The axillary nodes were larger than the cervical, varying from barely palpable nodules up to one or two centimetres in diameter. No history could be elicited of a wound or sore on the hand, arm or adjacent trunk which might be construed as a portal of entry. The tonsils were moderately enlarged and injected. The palpable glands in the neck were all in the supraclavicular triangle. None could be felt in the submaxillary region, though his stocky, thick neck could easily have caused one to overlook moderate enlargement of the deep glands. It was decided to have the tonsils removed, which was done together with some adenoid tissue October 10, 1924. Microscopical examination was not made. It seemed likely that such an exceptionally healthy appearing individual would be able to deal with the glandular infection after removal of the troublous primary focus.

He was readmitted to the Abington Hospital, January 27, 1927, slightly more than three years later. His general health had continued to be excellent. The cervical nodes had diminished in size and were entirely quiescent. The axillary nodes now formed a conglomerate mass as large as a lemon. The mass was removed *en bloc* and there has been no recurrence in this region.

Two years later he was readmitted with marked enlargement of the cervical glands on the right side, which had begun suddenly two months before. There were many glands in all the lateral triangles. They were tender and quite highly inflammatory in character. One in the post auricular area had evidently undergone partial softening. The general health was still excellent. The throat appeared normal. January 24, 1929, the glands were removed by what amounted to almost a bloc dissection. A part of the sternomastoid was involved and was removed. The spinal accessory nerve had to be dissected from an inflammatory mass which completely enveloped it. Fortunately, no paralysis resulted. Enlarged glands were present in the substance of the lower pole of the parotid gland which was resected. A temporary partial facial palsy followed which rapidly cleared up. Recovery was uneventful.

Four months later, May 12, 1929, he was again admitted on account of glandular swelling which had suddenly come on in the submaxillary triangle on the left side. Realizing from previous experience that spontaneous subsidence could not be expected a wide excision was made of the infected glands and of the adjacent lymphatic bearing tissue as well. There has been no recurrence on this side.

Three weeks ago a sudden enlargement began just below the mandible on the right side. This is in a spot which was spared in the former dissection because of danger of damaging the submaxillary branch of the facial nerve. Doctor Pfeiffer decided to try the effect of a few X-ray exposures on this single isolated gland. The diagnosis in this case has been verified by two guinea pig inoculations from glands removed at the second and third operations.

DOCTOR EIMAN expressed the opinion that the infection is probably of the bovine type. If so, this disease may have come originally through tonsillar infection from milk or butter.

DOCTOR PFEIFFER remarked that this patient is an example of a surgical condition, once common but now rather rare. The reasons for this decreased frequency are (1) the diminution in all forms of tuberculosis, (2) the sanitary precautions thrown about milk and dairy products, (3) the common removal of tonsils and adenoids which are the usual primary foci and (4) the idea that glandular tuberculosis, even if present, is not a surgical disease. It is well to sound a note, now and then, that this latter idea is not altogether correct. It is true that many cases of tuberculous lymphadenitis particularly in children, will regress, become symptom free and quiescent after removal of primary foci, and institution of the well-known hygienic measures directed towards the control of tuberculous infections. The use of the X-ray, ultraviolet light, tuberculin and local measures such as aspiration of softened glands have all enjoyed at times considerable popularity as adjuvants in treatment. The X-ray has established itself as a valuable agent in certain cases, ultraviolet light as well as X-rays are often successful in clearing up simple persistent tuberculous sinuses. Tuberculin has not fulfilled the early hopes. Aspiration of softened glands and injection of various substances has met with no favor in this country. Local applications and counterirritants have been found to hasten softening in many cases. In the multiplicity of measures available and at times successful, it would appear that the profession is not clear upon the relative values of the various methods. There can be no question that it is of paramount importance to remove the primary focus in all cases of glandular tuberculosis. In cervical adenitis this usually means tonsillectomy and adenoidectomy. Also, it is equally important to institute the recognized antituberculosis regimen. There can be no objection to ray or light therapy for a reasonable trial. It must not be overlooked, however, that not all glandular infections can be disposed of in this manner and one should be alert to note those which continue to progress or are lagging unduly under such measures. Dowd, long ago, showed incontestably that the removal of tuberculous cervical glands, while they are still confined to the earliest involved groups in the superior deep cervical chain is almost invariably followed by complete and permanent cure. Hanford more recently has confirmed this experience. This year, Miller and Shedden in reviewing the cases for the Massachusetts General Hospital have pointed out an additional reason for early removal of these glands. They were able to trace 132 of 204 cases and found that 29 (22 per cent.) had died of some form of tuberculosis. They quote the recent opinion of experts in tuberculosis that glandular tuberculosis is often associated with a mild bacteremia. The path is from the primary focus to the nodes, thence to lymphatic trunks and onwards to the venous system, then to the lungs and in many cases to the systemic circulation. While the incidence of glandular tuberculosis is greatly lessened at the present time, it is worth while to call attention again to the

serious results that often follow delay and neglect of these lesions and to remind the profession that in early surgical removal of primary lesions and primarily infected glands, we have the best insurance against local spread and systemic involvement.

Dr. John H. Jopson said that this case raised the question as to the point of infection. In tuberculous glands of the neck, of course, the point of entrance is generally in the throat, but in tuberculous lymphadenitis of the axilla and groin the hand or foot may be the site of the initial lesions. The speaker recalled a case which had come under his care in which axillary involvement was traced to an injury to the finger and the connection between the injury and the condition of the axilla was shown with sufficient clarity to convince an Indemnity Insurance Company. The patient had been engaged in unpacking pottery which was surrounded by straw; an injury to the finger by a thistle in the straw was the apparent portal of entrance for the tuberculosis. This man quickly developed a large mass of tuberculous glands in the axilla with mixed pyogenic infection which Doctor Jopson removed. The infection was probably of the bovine type. Doctor Jopson agreed with Doctor Pfeiffer that these cases were best treated by surgery which as C. M. Dowd has shown will cure 85 per cent. of the cases in one operation. At least in children. Although tuberculous lymphadenitis is not seen nor discussed with anything like the frequency of a number of years ago, Doctor Jopson remarked that it was still very common in the colored race and that in his service at the Graduate Hospital, where a large number of colored patients are treated, he still sees the very worst type of cases.

Dr. George M. Dorrance said that in his experience years ago surgery alone had been extremely unsatisfactory. He has had excellent results since using X-ray and surgery and X-ray combined with local removals and not radical excisions. It is taken for granted that these patients must have all foci of infection in the mouth removed and must be treated with sunshine, fresh air and forced feeding the same as any other tuberculous patient. The speaker did not believe that the results, including a certain mortality, justify radical excision.

Dr. John H. Jopson said that this question of the treatment of cases of tuberculous lymphadenitis in the neck especially is an old quarrel between Doctor Dorrance and himself. The fewer of them are being operated upon because fewer cases occur. The same is true in London where the present statistics show a marked reduction of the number of such cases observed and operated upon. This is apparently due to the fact that the milk supply of the city of London has been cleaned up and fewer patients become infected with the bovine type of tuberculosis. Doctor Dorrance's optimism is hard to understand, especially in view of the fact that one finds no such attitude in consulting the best X-ray men of this city. Doctor Jopson reiterated his position by stating that he believed the condition to be essentially a surgical one in the stage in which the majority of cases where encountered and that operation was justified and indicated.

## THE NATURAL CHEMICAL STIMULUS FOR CELL DIVISION

DR. F. S. HAMMETT, by invitation, remarked that a fundamental discovery would be that of the chemical differences between a cell in mitosis and one resting. The work at the Research Institute of the Lankenau Hospital began with this problem. It was known that lead would retard cell division, from the work of Blair Bell and his assistants. How it did, remained to be investigated. Growing root tips of onions, corn and beans in standard cultures containing a small amount of lead nitrate showed that the lead was precipitated in the region of active cell division and that further, this precipitate was very much more abundant within the nucleus than at any other place. Chemical analysis under the microscope brought out the fact that this was a compound of lead with the organic radicle —SH. Numerous compounds were tested in pairs, one containing —SH and the other exactly similar except that another radicle, usually —OH, was contained within its molecule instead of —SH. It was found that the compounds containing the available —SH group stimulated mitosis in plants, in paramecia and in rats. Experiments with the latter were done in this way: Two equal-sized pieces of skin were excised from a rat, one on each side of the flanks. Thio-glucose was placed on the right wound and glucose on the left. Those wounds treated with thio-glucose healed more rapidly than the others. That is to say, the thio-compound stimulated cell division to healing. The use of this radicle in man was then undertaken with the results as outlined below.

These results are to be found in detail in "Protoplasma," 1928:1929, and the Journal of Experimental Medicine, 1929.

## THE USE OF THE NATURAL CHEMICAL STIMULUS FOR CELL DIVISION IN WOUND HEALING

DR. STANLEY P. REIMANN, by invitation, said that a colored man, seventy-eight years old, with an ordinary varicose leg ulcer of twelve or more years' duration was first treated. One-half of the wound was flooded with thio-glucose; the other half was painted with mercurochrome. At the end of twenty-four hours about two square inches of epithelium had grown on the thio-treated part. No progress was made on the other half. This wound healed in two weeks sufficient to allow the application of a plaster case for a fractured thigh for which the patient had been admitted to the hospital.

Similar experiences were encountered in several other leg ulcers and in several bed sores.

The thio-glucose, as prepared by Doctor Toennies, Organic Chemist to the Institute, was used in a one to ten thousand slightly acid solution and applied as a wet dressing.

There can be no doubt that these thio-compounds stimulate the rate of cell division. But much further clinical experimentation will be necessary to determine the most advantageous compound to use; whether it is better to use as a wet dressing, or by the drip method, to use it constantly or interruptedly, etc.

Thio-glucose also stimulates bacterial growth and it was thought that perhaps coupling the thio-radicle to a substance like phenol or cresol would overcome this objection. Later experiments, using thio-cresol, bore out this idea, for several leg ulcers and a bed sore treated with these substances showed a minimum amount of surface pus. Once again, however, the most advantageous concentrations and means of application require further investigation. For further details of this work see 1929, Reimann, S. P., and Hammett, F. S., Proc. Soc. Exp. Biol. & Med., vol. xxvii, pp. 20–22.

Dr. Astley P. C. Ashhurst confessed to more or less ignorance, regarding SH and OH groups, etc., but said that he was aware of the fact that rest in bed, elevation and strapping with adhesive plaster would cure leg ulcers.

### STATED MEETING HELD DECEMBER 2, 1929

## HÆMOLYTIC ICTERUS; SPLENOMEGALY, MULTIPLE ABSCESSES OF SPLEEN, SPLENECTOMY

Dr. Norman S. Rothschild presented a young woman, aged twenty years, who was admitted to the Northern Liberties Hospital, in the service of Dr. Leonard Averett, September 14, 1928, with the history of having had a three months' pregnancy interrupted six days before. This condition was accompanied by severe hæmorrhage. The patient complained of severe pain in the left lower abdomen and pelvic examination revealed a uterus somewhat larger than normal, enlargement being due to subinvolution and not to retained products of conception. The left tube was enlarged and tender; the involvement of the right was the same, but to a lesser degree. The abdomen was not distended; there were visible pulsations. Liver dulness was increased about two inches below the costal margin. The spleen was palpable. A soft systolic murmur was transmitted to the vessels of the neck and to the axilla. The heart sounds were weak. Her skin was greenish yellow in color. Temperature was 103 2/5, pulse 118, respirations 28. Blood pressure, 100 over 60. The blood count was erythrocytes 1,700,000, hæmoglobin 28 per cent. and 8,100 leucocytes. The urine showed albumen and twenty erythrocytes to the field.

Her previous medical history was very interesting. She stated that she had been a patient in the Children's Homeopathic Hospital two years ago, suffering with jaundice, anæmia and an enlarged spleen. This jaundice was noted by her sisters and brothers for many years and she was constantly the subject of teasing because of her yellow color. A report from the hospital stated that she was admitted with a severe anæmia, jaundice, and an enlarged spleen, which extended into the left iliac fossa. Her blood count at that time was as low as 2,200,000 erythrocytes and 48 per cent. hæmoglobin. The Van den Bergh was direct; delayed and indirect; bilirubin slightly positive. Fragility of red corpuscles, complete hæmolysis in 0.44 per cent. salt solution.

The following day she was given 200 cubic centimetres of blood and showed a slight improvement. The temperature began to approach normal and 400 cubic centimetres of blood was given two days later. Eleven days after admission her temperature rose to 104, then to 104 4/5 with some remission and then to 106. Her pulse and respirations increased with the temperature rise. She was again transfused, 300 cubic centimetres of blood being given. The transfusions were not attended by reactions. Blood cultures were sterile. The blood counts showed but a slight increase in the erythro-

cytes and hæmoglobin from the transfusions. The icteric index at first 9, rose to 60. The Van den Bergh at first delayed direct, became moderate direct. A firm mass, tender to touch, developed in the left lumbar region. Possible kidney involvement was considered and a pyelogram was made. X-ray findings of Doctor Bruck were as follows: "A large dense mass is seen in the left abdomen reaching down to the crest of the ilium and a little below it which merges in its upper portion with the shadow of the spleen. It is too far external to be the kidney." The jaundice increased and there was considerable tenderness over the gall-bladder. Cholecystographic studies showed that the gall-bladder was not definitely outlined. No dye stained shadows of stones were seen and after the ingestion of food, there was no change in the appearance. The conclusions were that the failure of the gall-bladder to outline in spite of the absorption of most of the dye, was suggestive of a chronic gall-bladder condition with obstruction in the cystic duct.

Doctor Rothschild saw the patient eighteen days after admission, and because of the enlarged spleen and the history of its enlargement two years before, the character of the temperature, which was "pump handle," the history of the pelvic infection and the general condition of the patient, felt the condition to be a blood stream infection despite negative blood cultures, and recommended the use of Pregl's iodine or mercurophan intravenously. Dr. S. A. Lowenberg saw the patient the same day and made the following report: "Because of previous history of enlarged spleen and anæmia, both of which have become aggravated with the acute infection, it is plausible to consider that she has a chronic splenomegaly superseded by an acute infectious splenitis." He recommended X-ray treatments. Intravenous therapy and X-ray treatments had no affect upon the patient. Biliary drainage was performed with good results, the jaundice being less. This procedure was performed every day with some relief to the patient. The jaundice, however, varied at times. Numerous attacks of severe sharp pain in the left lumbar region occurred and these were interpreted as emboli of the spleen. The bleeding time was three and one-half minutes and the coagulation time was five and one-half minutes. Repeated pelvic examination showed these organs to have returned to the normal size.

The patient's general condition did not improve, and on December 4, 1928, splenectomy was performed. The spleen was adherent to the diaphragm, to the stomach, transverse colon and descending colon. The diaphragmatic adhesions were separated with considerable difficulty, but without hæmorrhage. The other adhesions were severed between ligatures. As the pedicle was brought into the wound there was spill of pus from the spleen. The vessels were doubly ligated and severed. No accessory spleens were seen. Because of the spill of pus the gall-bladder and liver were not inspected. The wound was closed in layers and drainage was instituted through a stab wound posteriorly. The patient's condition was very poor on the table and stimulation was resorted to. Her post-operative course was uneventful with the exception of an occasional rise of temperature to 101 to 102 and once to 103, then to a return to normal. The greenish yellow hue which this patient exhibited before operation gradually faded. The drainage was at first sanguineous but later became purulent. Slight tenderness over the gall-bladder was present at times, but duodenal lavage would relieve it.

February 19, 1929, she was readmitted complaining of a pain in the right and left upper quadrants and considerably jaundiced. She had a sinus in the abdominal wound from which greenish pus was discharging. There was tenderness and rigidity of the abdominal muscles in the upper quadrants.

The temperature and pulse were normal. Blood count showed 3,410,000 erythrocytes and 57 per cent. hæmoglobin. Icterus index 33, Van der Bergh direct immediate. Biliary drainage showed "A" bile only. Temperature remained normal about a week when she became toxic, temperature was elevated and she developed pain and tenderness in the left upper quadrant. The sinus was investigated under gas anæsthesia and a small amount of greenish pus exuded. Drainage was established. The temperature remained high for four days and then became normal until the time of her discharge from the hospital.

Seven months after operation she still complained of slight pain in the upper right quadrant and was receiving treatment in the gastro-intestinal clinic.

The pathological examination of the spleen was as follows. It measured 36x15x5 centimetres. The capsule appeared normal and on sectioning there was an abscess 5 centimetres in diameter, with a rough gray wall. There were several small abscesses scattered throughout. There were numerous yellow areas, which are firmer in consistency and are infarcts. The culture for this abscess showed pure colon bacilli.

*Post-Operative Blood Studies Showed the Following Change in the Blood Picture*

| Time | Erythrocytes | Leucocytes | Hæmoglobin per cent. |
|---|---|---|---|
| 3 days | 2,330,000 | | 40 |
| 4 days | 2,790,000 | | 42 |
| 6 days | 2,990,000 | | 40 |
| 16 days | 2,990,000 | | 42 |
| 18 days | 3,030,000 | 25,500 | 47 |
| 49 days | 3,400,000 | 15,600 | |
| 10 weeks | 3,410,000 | | 57 |
| 7 months | 4,500,000 | 12,800 | 75 |

The speaker remarked that Billings in his paper on Abscess of the Spleen reported three cases, two from blood stream infections. That of Doctor Jopson's from puerperal sepsis, his own from a carbuncle of the neck, and that of Doctor Klopp from a urinary tract infection. He states that as the evolution of the abscess progresses from the upper pole towards the thorax, or from the lower pole towards the general peritoneum, symptoms of a pleuro-pneumonia or abdominal nature may develop. The treatment of uncomplicated cases, is splenectomy, of the complicated, splenotomy. In this case splenectomy was performed without the knowledge of the existing abscesses, although the chief resident physician, Doctor Zimoring, believed this condition existed. Without splenectomy this patient's primary condition, hæmolytic icterus, would certainly have not been cured.

## PERFORATED DUODENAL ULCER WITH MULTIPLE SEQUELÆ

Dr. Edward J. Klopp reported the case of a man, twenty-seven years of age, who was admitted to the Memorial Hospital, September 10, 1928, with the diagnosis of perforated duodenal ulcer. He had had fairly definite ulcer symptoms for three years. The attacks were subject to seasonal exacerbations occurring most often in the fall and winter, subsiding during hot weather and would last for from five to eighteen weeks and disappear for a similar period. Two days prior to admission, three hours after breakfast, he complained of nausea and took no more food that day. The ulcer perforated on day of admission, five hours after breakfast, and he was

operated upon eight hours after perforation under nitrous oxid and oxygen. There was a moderate amount of turbid fluid in the peritoneal cavity. The ulcer was closed with a linen suture. A typical posterior gastro-jejunostomy was done using linen for the serous suture. A rubber covered gauze drain was inserted to the subhepatic space. Smears and cultures of the peritoneal fluid showed streptococci and staphylococci. Convalescence was satisfactory until the twelfth day when he had pain in the right lower chest and a temperature of 102. X-ray of the chest on October 6, 1928, revealed no evidence of a lesion above the diaphragm. The diaphragm was in normal position, but slightly restricted from its excursion. October 13, 1928, a right subphrenic abscess was drained by resecting a portion of the eleventh rib under local anæsthesia. Culture showed streptocci and staphylocci. October 19, there was X-ray evidence of right pleural effusion and an interlobar shadow suggestive of an abscess. He coughed considerably, continued to lose weight and his temperature was of the septic type. The symptoms and signs indicated a large collection of fluid in the right pleural cavity. X-ray on November 7, 1928, seemed to show less fluid than at the previous examination. November 18, 1928, the right chest was drained of a large quantity of foul smelling pus by resecting a portion of the ninth rib under local anæsthesia. Culture of pus obtained four days previously by aspiration of the chest showed large rods, most likely colon bacilli. At a subsequent X-ray examination a piece of rubber tubing three inches in length was found within the pleural sac. It was easily extracted with forceps. He had improved sufficiently to leave the hospital December 22, with pus draining from the pleural cavity and from the abdominal sinus. The wound to the subphrenic space had closed.

February 11, 1929, while a nurse was irrigating the abdominal wound with salt solution the patient coughed up the irrigating fluid and a considerable quantity of pus. He was admitted to the Pennsylvania Hospital two days later. Lipiodol was injected through a catheter in the abdominal sinus. It followed underneath the dome of the diaphragm to about its apex, at which point a very small shadow of lipiodol seemed to pierce the diaphragm and came to an abrupt end. They had expected to demonstrate a fistula between a bronchus and the abdominal sinus communicating through the diaphragm. Irrigation of the empyema cavity distinctly showed it to communicate with a bronchus. Lipiodol injected through the empyema wound showed a cavity of 2 or 3 cubic centimetres. He was discharged from the hospital, March 9, 1929, with no clinical evidence of bronchial fistula and the abdominal wound was healed.

April 8, 1929, he complained of severe headache which was continuous and was readmitted April 11, in a stuporous condition, moaning and mumbling of pain in the head. He did not move his left extremities. The neurologists consulted thought he had a brain abscess deep in the right frontal region with secondary meningitis. They advised against operation and the patient rapidly went into coma and died on April 14. Blood culture was negative. Spinal fluid examination showed a great many pus cells; no organisms. Culture showed no growth within forty-eight hours.

At autopsy there were adhesions extending from the incision backward and upward to the liver; no free fluid. The opening between the stomach was large, free of scars or adhesions. The mucosal surface of the ulcer showed it to be fairly well healed. No scars or adhesions were found about it except for the scar of the ulcer itself. A small portion of linen suture remained on the serosal surface. The upper and middle lobes of the right lung were rather dark and mostly air-containing. In the lower lobe a

bronchus extended into an area consisting of a central abscess of about 2-3 cubic centimeters in size with five or six smaller abscesses scattered near it. Adhesions obliterated the interlobar fissures quite well. There was no empyema cavity that could be distinguished. In the diaphragm there was no path or scar to indicate direct extension of pus from below upward. *Brain and skull.*—On exposing the skull just above the right motor area there was a superficial staining with blood. On removing the skull cap no evidence of deep lesion was found in the bone. The dura contained moderately injected vessels. On incision in the midline just anterior to the central portion of the brain considerable amount of dirty gray pus exuded on the slightest pressure. There was evidence of meningitis over the vertex. The medulla, pons and part of the cerebellum adjoining them were covered with a thick, yellow plastic exudate possibly greater on the right side. The culture showed colon bacilli and staphylocci. The abscess was later reported to have been in the frontal and temporal lobes.

The reporter stated that he had lost three patients with perforated duodenal ulcer in which there was subphrenic infection. In one of these a drain had been placed between the liver and diaphragm. He developed empyema. Autopsy showed a necrotic area in the diaphragm 3 centimetres in diameter with perforation. Another case, not his own, died three days after the operation for perforated ulcer, from respiratory infection. Autopsy also showed a gangrenous patch in the diaphragm without perforation. The peritoneum was wiped fairly dry in the case reported. It is difficult to prevent subphrenic infections in these cases.

## RUPTURED ADENOMA OF THE THYROID

Dr. Henry F. Ulrich, by invitation, reported the case history of a man forty-two years of age who was admitted in the service of Dr. Charles H. Frazier at the University of Pennsylvania Hospital July 11, 1929. His chief complaints were: difficult breathing which appeared suddenly, and with swelling of and pain in the neck. For eight years previous to admission he had had an enlargement of the right lobe of his thyroid which from his history was undoubtedly a toxic adenoma. About three hours before his admission to the hospital, while having a friendly tussle with a friend, he suddenly developed a sense of suffocation, was unable to speak and could breathe only with great difficulty. Immediately he developed severe pain in the base of his neck as the neck became swollen and tense. Severe dyspnœa persisted for about one hour, but was considerably relieved at the time of admission. By that time, however, the swelling had increased and dysphagia became a troublesome complaint.

His past medical history and family history were negative.

He was a well-developed large man. Blood pressure 140/70; temperature 99.4; pulse 100; respirations 22. The face was flushed and slightly dusky. Eyes, ears, nose and mouth were grossly negative. Neck showed a tense infiltrating swelling of almost the entire anterior and lateral aspect, more marked on the right side. There was no discoloration or œdema of the skin and the mass was only slightly tender. Heart and lungs were essentially negative.

Dr. I. S. Ravdin saw the patient soon after admission and made a diagnosis of ruptured adenoma of the thyroid with hæmorrhage into the neck. As the patient was in no eminent danger from tracheal compression, operative intervention was voluntarily delayed. The patient was given a hypodermic of morphine sulphate grains 1/6 and atropine sulphate grains

1/150 and an ice-collar was applied. He had a fairly comfortable night. The following day Dr. Gabriel Tucker examined the patient and reported as follows: "Examination of the pharynx shows considerable bulging on the right side of the posterior pharyngeal wall. The mucosa is bluish in appearance. Acinitis and discoloration extends around the right lateral wall of the pharynx, the base of the tongue, and the epiglottis. There is considerable swelling in the pyriform sinuses on either side, apparently due to swelling of the posterior and lateral pharyngeal walls. Larynx—motility normal, although there is considerable displacement of the larynx toward the left with tilting toward the left side. Both sides seem to move equally. No evidence of compression of the trachea on mirror examination. This, of course, does not exclude the lower portion of the trachea, as only the upper two or three rings could be seen with the mirror. Patient was not dyspnœic at the time of examination. This condition could arise from a submucous hæmorrhage into the tissues of the pharynx extending around the lateral and posterior walls.

During this day there was gradual but increasing dyspnœa until 5.30 P. M. when the patient was operated upon under local anæsthesia by Doctors Frazier and Ravdin. A mass consisting of ruptured adenoma and blood clot the size of a small grapefruit was found. The blood had infiltrated all the muscles of the neck including the pharyngeal wall. The sterno-cleidomastoid muscle, which has its own sheath, was also infiltrated and clots filled the retrotracheal space from the level of the sterno-clavicular articulation to the mandible. There was some infiltration of blood on the left side. A right lobectomy was done, the wound packed with iodoformed and vaseline gauze, and left open. Hæmostasis was complete at the close of the operation. The post-operative convalescence was uninterrupted until June 21, when a secondary closure of the wound was done. June 28 the patient was discharged with the wound still draining some serum. July 4 the discharge from the wound became purulent and the patient said his neck became painful. On July 13 he was readmitted to the hospital with a marked cellulitis of his neck. At this admission he had some dysphagia but no dyspnœa. Temperature was 102, pulse 98. The incision was reopened under local anæsthesia that evening and packed with iodoformed gauze. Moist heat was kept on the wound and at the end of twenty-four hours his temperature and pulse were normal, and remained so until discharge on the ninth day. During this admission he was again examined by the Bronchoscopic Service who reported the pharynx and larynx normal.

Six weeks later the patient reported that his wound was healed and that he was doing his usual work with no discomfort.

Primary emergency surgery of the thyroid gland is decidedly unusual. The outstanding indication for such is hæmorrhage, either into the gland or from the gland into the neck, of sufficient amount as to cause respiratory embarrassment. Hæmorrhage may be spontaneous or the result of trauma. There is considerable variety in the degree of hæmorrhage encountered, from that reported by the pathologist, in specimens of small adenomas with cystic changes to the massive hæmorrhage causing such tracheal compression that death would ensue without prompt intervention.

The largest series of cases of spontaneous hæmorrhage into a goitre which has been published is that reported by Schwoerer [1] who found 18 such cases among 2,500 goitre patients, with a mortality of 27.7 per cent. Von Ziemack [2]

reported one case in which the subcutaneous extravasation spread down the chest and abdominal wall as far as the umbilicus. Haim [3] reported several cases of spontaneous hæmorrhage into strumous thyroids. Deegan [4] has recently reported a case which is strikingly like ours.

Gunshot and stab wounds were responsible for the traumatic cases reported by Alamartine [5] and Lenormant.[6]

The diagnosis of rupture of an adenoma of the thyroid with hæmorrhage into the neck should not be difficult, especially when history of preceding goitre is obtained. A history of sudden increase in the size of the neck with phenomena of compression makes it a reasonable supposition. The symptoms of hæmorrhage reach a climax and subside more quickly than in acute inflammation of the thyroid (Crotti).[7]

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[4] Deegan, J. K.: Clifton M. Bull., vol. xv, p. 85, 1929.
[5] Alamartine, H.: Presse. Med., vol. xxvii, p. 107, 1919.
[6] Lenormant, C.: Presse. Med., vol. xxxvi, p. 1919, 1928.
[7] Crotti, Andre: "The Thyroid and Thymus," Lea and Febiger, Phila., 2nd ed. p. 108, 1922.

PHRENIC NERVE STIMULATION IN DIAPHRAGMATIC HERNIA

Dr. Richard Overholt, by invitation, read a paper with the above title for which see page 381.

Dr. George P. Muller said that he wished to emphasize the point brought out by Doctor Overholt regarding the excellent relaxation of the abdominal wall by spinal anæsthesia and of the diaphragm by the phrenic nerve freezing. Often in the literature, the old approach is condemned because of the difficulty of operation, but these procedures made operation exceedingly easy. The speaker was disappointed with the recurrence, but would be willing to operate again and use silk sutures. The suturing was very carefully done with chromic catgut mattress sutures, overlaid with staggered interrupted sutures. Doctor Overholt and he had read Harrington's article, but were not aware that the preliminary stimulation of the phrenic nerve had been done before as an aid to diagnosis. This was Doctor Overholt's suggestion and it worked out quite well.

MORTALITY AND END RESULTS OF OPERATION FOR ABSCESS OF THE LUNG

Dr. George P. Muller read a paper with the above title for which see page 361.

# TRANSACTIONS

OF THE

# NEW YORK SURGICAL SOCIETY

STATED MEETING HELD DECEMBER 11, 1929

The President, Dr. Edwin Beer, in the Chair

## PHRENIC NERVE RESECTION FOR LOWER LOBE BRONCHIECTASIS

Dr. Ira Cohen presented a man, thirty-six years of age, who in July of this year was admitted to Mt. Sinai Hospital, stating that two years before, he had had a tonsillectomy performed; about three weeks after which he had pain in the left chest posteriorly and a heavy feeling in the anterior part of the chest, with chills, fever up to 105°, and a cough with profuse foul sputum. This episode lasted five weeks and then all symptoms disappeared. For more than a year he remained well. In October of last year, he had an acute respiratory infection which lasted a week and was followed by a persistent cough with foul sputum. This subsided in part but he was never free. Four weeks prior to his admission to the hospital he began to run fever up to 102°. Sputum became profuse and he suffered again from pain in the left chest. A week prior to admission, he had a hæmoptysis after which his temperature became normal but the cough, sputum and pain persisted. He had lost eighteen pounds in weight in two years and for a month was too weak to work.

The patient looked ill. He had some slight curving of finger nails. There was dulness with diminished breath and voice sounds at the left base. His hæmoglobin was 71 per cent. All other laboratory tests except X-ray were negative. The X-ray of the chest showed irregular infiltration of the left lower lobe with pleural and pericardial adhesions.

He was bronchoscoped by the laryngological department and the middle branch of the left lower lobe bronchus was found dilated and pus was seen coming from it. Iodized oil was injected into the trachea and the patient was X-rayed. This plate brought out the small bronchiectatic cavities and dilated small bronchi. It was felt that he would be improved by compression of the lower lobe obtained by means of a phrenic nerve resection. This was done August 13. Two days later fluoroscopy and X-ray showed the elevation of the left side of the diaphragm. He left the hospital five days after the operation. He has improved and has returned to work. Occasionally in the morning he coughs and brings up a teaspoonful of sputum which at times has an odor. He has also a sense of fullness in the front of the chest. The patient is being shown improved, though not cured, following his operation. As the lung involvement was not widespread and was confined to the lower lobe, he seemed to be a particularly favorable case for this type of operation.

Doctor Cohen presented this patient not as a cured but as an improved case. An interesting fact in connection with it was that on the morning following the operation the patient volunteered the information that his cough was easier and that the expectoration had diminished.

Dr. Howard Lilienthal said he had done several phrenic nerve resections for this purpose and so far had not succeeded in curing a patient, in spite of the fact that there was complete paralysis of the diaphragm on the affected side. These patients have remissions, and he believed this patient had had a remission although he hoped that the present one will prove to be not a remission but a permanent cure. It was to be hoped that he will be presented again in say one year and again in five years to see if he remains well. On examining these cases with the X-ray it is important not only to fluoroscope them but to take a film on deep inspiration and one in deep expiration so as to have a record of the rise of the diaphragm. Another important point was that in performing artificial pneumothorax it must not be forgotten that this will push the diaphragm down, sometimes to a great degree. This should be borne in mind by those doing operations of this character so they will not trust entirely to what they see in the röntgenograms.

## FASCIAL SARCOMA OF THIGH

Dr. Ira Cohen presented a man who in March, 1921, when forty years of age, was admitted to Mt. Sinai Hospital because of swelling of his left thigh. This had begun six years previously as a small painless lump above the knee. Two years later it began to grow slowly and gave him some pain. For two years prior to admission the growth had been more rapid. He had lost twenty pounds in weight in these two years. He had a persistent cough which at times was productive. He had not had any hæmoptysis or fever. There was a history of a chancre some twenty years ago. Aside from the local condition the only positive physical finding was unequal pupils which failed to react to light. Laboratory tests including Wassermann were negative. On the anterior surface of the left thigh, about in the centre, was a hemispherical tumor which measured twenty-two centimetres from above down and from side to side. The left thigh measured fifty-one centimetres in circumference at the most prominent part of the swelling—the right thigh, thirty-seven centimetres at a corresponding point. The skin over the tumor was bluish in color. The mass itself seemed somewhat cystic. It was not attached to the bone, and seemed to be drawn up into the quadriceps when it contracted.

With a pre-operative diagnosis of sarcoma, operation was performed March 10, 1921. A small probatory incision revealed necrotic tumor. An elliptical incision was made through the skin. The muscles were divided well beyond the growth. The femur and femoral vessels were laid bare. After removal of the growth, tissue which macroscopically seemed involved was excised from along the course of the femoral vessels. The muscles sacrificed were practically all the components of the quadriceps femoris from six centimetres above the patella upward to ten centimetres from Poupart's ligament. Part of the tensor fasciæ femoris and most of the sartorius were also sacrificed. Closure was made by suturing the tensor fasciæ femoris to the adductor group. A tube drain was inserted into the dead space after suturing the skin. The specimen, which was the size of a small football, was made up of muscle and fascia infiltrated with tumor tissue in part necrotic. There were signs of a hæmorrhage into the upper part. The report from the pathologist was spindle-cell sarcoma. The patient was discharged on the

nineteenth post-operative day. A small superficial sinus remained. He was referred to the department of radiotherapy for X-ray treatment.

Ten months later, in January, 1922, he was sent into the hospital again from the follow-up clinic because of a cough with blood-streaked sputum and pain in the right axilla and dorsal spine which he had noticed for six weeks. He claimed to have lost ten pounds in weight. A small nodule was seen in the left supra-patellar region. There was dulness with diminished breath and voice over the left upper lobe. X-ray examination of the chest showed an extensive pleuritic process of both bases. The nodule in the scar was excised and reported chronic inflammatory tissue. This time his Wassermann reaction was 4 plus.

The patient was lost sight of for some years. Recently, he reappeared because of hemorrhoids. He was now presented as a late result following excision of an extensive sarcoma, and because, in spite of the sacrifice of considerable muscle, the function of leg is very little impaired.

Dr. Frederic W. Bancroft raised the question whether, in reviewing the case, the original microscopic diagnosis of sarcoma should not have been gumma.

Doctor Cohen replied that the same thought had occurred to him, as the result of which he went to the pathological laboratory to have the slides reviewed. At the time the patient was operated on, in 1921, Doctor Mandelbaum was pathologist at the Mt. Sinai Hospital. The present pathologist, Doctor Klemperer, examined the slides. The first one looked at, he called a gumma but this slide was made from the nodule which had been removed from the scar at the second operation. The original slides were finally found and Doctor Klemperer said, "This is undoubtedly sarcoma."

## CARCINOMA OF REMAINING BREAST FOLLOWING RADICAL MASTECTOMY

Dr. Ira Cohen presented a woman who in June, 1924, when forty years of age, noted a lump in her right breast, which she states developed following a blow from the corner of a food truck. She failed to report the presence of this tumor until January, 1925, at which date there was a hard fixed tumor in the outer half of the right breast with obvious axillary node involvement. She was given one pre-operative X-ray treatment and a radical mastectomy was done. The pathological report was medullary carcinoma with metastases to the axillary nodes. Following the operation, she was given X-ray treatment. In August, 1925, while under treatment, a small nodule appeared in the left supraclavicular region which disappeared.

In October, 1926, one year and nine months after the first operation, in the course of a routine follow-up examination, a small tumor was felt in the left breast, below the nipple. There was some retraction of the nipple. No axillary nodes could be felt. X-ray examinations of spine, pelvis and lungs were negative for metastases. A report from a frozen section done at time of operation, November 9, 1926, was carcinoma. Radical removal of the left breast was then performed. As in the first breast, the specimen was reported medullary carcinoma with involvement of one axillary node. She had further X-ray treatments after the operation, none since early in 1927. It is nearly five years since her first operation and more than three years since the second. She has remained well. Recent examination fails to disclose any metastases.

Involvement of a remaining breast by carcinoma after radical mastectomy while not frequent is not excessively rare. The second carcinoma is usually considered a metastasis. It is of interest that in this instance, as in some other cases reported, removal of the second breast is followed by freedom from further disease over long periods of time. Although the carcinoma in the second breast is usually considered a metastasis, the fact that no other metastases are noted and that the patient is often cured by mastectomy leaves the question open as to whether in some instances, one is not dealing with a second primary tumor.

Dr. Howard Lilienthal agreed that this is probably a second primary carcinoma. A number of years ago he wrote a paper on the tendency to carcinoma and mentioned the case of a man who had carcinoma of the transverse colon which was resected and nineteen years later he came for treatment for carcinoma of the sigmoid. It was inconceivable that one had anything to do with the other. All surgeons have had cases in which carcinoma appears simultaneously in more than one part of the body.

## FRACTURE ABOUT ANKLE-JOINT: LATE OPERATION WITH RESULT

Dr. James M. Hitzrot presented a man, aged sixty, who broke his right ankle December 24, 1927. This was reduced under anæsthesia at a hospital and the leg put up in a circular plaster splint. His leg was kept in the plaster splint for six weeks and then removed and he walked on crutches for three months. After the removal of this splint he could not get his heel to the ground and walked about on his toes with considerable difficulty, and was able to walk only very short distances, about one block, and could stand for only a few minutes at a time. After use his leg swelled and became quite painful at the ankle-joint.

He was seen first by the reporter on October 29, 1928, approximately ten months after the injury. His right ankle was markedly deformed with the foot in the equinus position. The lower end of the tibia projected forward at the ankle-joint and the whole foot was displaced posteriorly and the heel was on a level with the projecting anterior portion of the tibia. The X-ray (Fig. 1) shows the lesion better than it can be described.

November 14, 1928, Doctor Hitzrot operated upon him, intending to do the operation, for old unreduced displacement, described by Stimson (*Fractures and Dislocations*, Phila., 1917, p. 463) which he had used successfully in old unreduced fractures, adding to it the lengthening of the tendo Achillis advocated by Dowd (Annals of Surgery, vol. lxviii, No. 3, Sept., 1918, p. 330) for recent fractures of the particular type to which this fracture belongs.

A four-inch incision was made, beginning on the right fibula and extending down the outer side of the leg to the dorsum of the foot. The site of the fracture in the fibula was exposed and the malleolus found to point posteriorly. So much new bone had formed that it was impossible to determine definitely the line of fracture. The fibula was divided in what was considered the line of fracture and the lower fragment mobilized. An internal incision was then made with its center over the lower end of the tibia and the lower end of the tibia exposed. The posterior portion of the tibia, which had been displaced backward, was then divided by an osteotome. This mobilized the lower end of the tibia which was then delivered through the wound on the inside of the leg. The scar tissue and new bone about the astragalus was then removed. A portion of the external face of the

articular surface of the astragalus had been eroded, and the articular surface of the astragalus was distinctly altered in shape. The tendo Achillis was so contracted that it was not possible to bring the astragalus forward. An incision was then made over the tendon and the tendon divided so that it could be lengthened, leaving the two long ends for subsequent approximation. When this was done it was perfectly obvious that the contraction of the soft tissues on the back of the leg and of the skin made the reduction of the displacement of the astragalus impossible, that is, the restoration of the normal ankle-joint was impossible. Any attempt to pull the foot down caused an immediate cessation of pulsation in the vessels in the foot.

The articular surface of the tibia was then projected from the wound and approximately one centimetre of the lower end of the tibia was removed with the saw. This left a broad surface which was reshaped so that it somewhat resembled the normal articular surface of the tibia. The astragalus was then placed in contact with the tibia and brought well forward and apparently established a satisfactory weight-bearing surface with the newly formed articular surface of the tibia. The under surface of the tibia was then smoothed off leaving a ridge on the inner side to form a new internal malleolus. A piece of bone was then removed from the fibula and the fracture line in the fibula reconstructed so that the course of the fibula was reestablished to form the external mortise of the ankle-joint. The reflected ligaments and anterior portion of the periosteum over the tibia were then sutured, reconstructing the anterior and internal portion of the ankle-joint. The external portion was also sutured in the same way, partially reconstructing the ankle-joint, especially on its internal and external aspects. The superficial wound was closed without drainage, using interrupted silkworm gut throughout. At the completion of these steps the astragalus apparently remained in position without any tendency to drop backward.

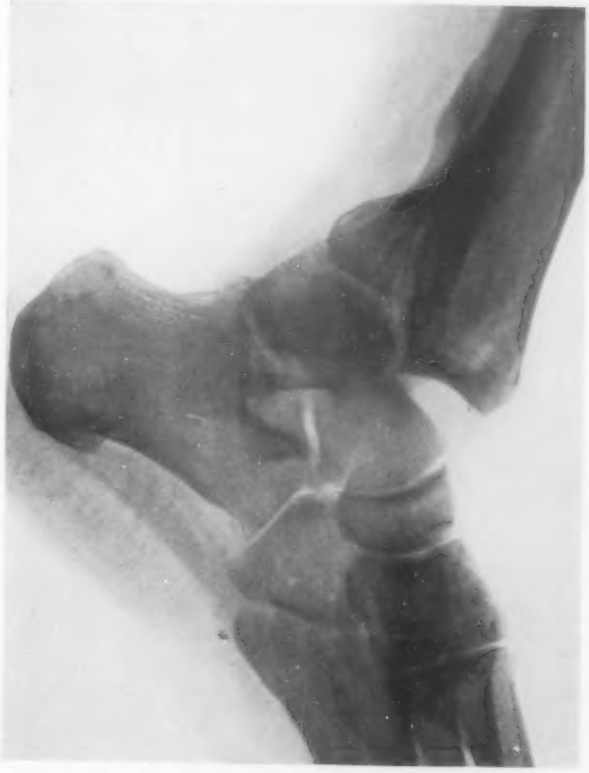

FIG. 1.—Fracture at ankle; condition ten months after injury.

The divided halves of the tendo Achillis were then approximated, lengthening the tendon about five centimetres, and suturing the tendon in its lengthened shape under sufficient tension to maintain a very slight pull on its attachment, but not sufficient to tend to pull the astragalus backward. This superficial wound was also closed with interrupted silkworm gut. The foot was dressed with thymol-alcohol dressings and put up in moulded plaster

splints posterior and internal lateral, from the mid-thigh to the toes with the foot in adduction and slight inversion of the sole with dorsal flexion a little beyond a right angle.

Note.—So many secondary changes had taken place in the lower end of the tibia and on the articular surface of the astragalus that it was impossible to restore the normal joint. The contraction of the soft tissues of the back of the joint likewise made this correction impossible. Figs. 2 and 3 show the condition after operation.

He made an uneventful recovery. His splints were removed at the end of six weeks. Massage and baking were begun on the fourteenth day and the massage was continued for approximately six months. Motion at the ankle was begun on the twenty-first day. He discarded his crutches seven weeks after the operation and the walking splint sixteen weeks after the operation. His recovery of muscle power was slow and is still impaired, but he can now walk without difficulty and has a fair range of motion at the ankle-joint in both dorsal and plantar flexion. Doctor Hitzrot presented

FIG. 2.—Fracture at ankle; condition after operation—antero-posterior view.

FIG. 3.—Fracture at ankle; condition after operation—lateral view.

the case to illustrate: first, the danger which may result from the slipping of a corrected displacement inside a plaster splint, as described by Dowd; second, the failure of the ordinary reconstructive operation to meet the conditions; third, an operative procedure to meet the conditions found. So far as he knew no similar procedure has been previously reported.

The result has been so gratifying in this case that he believed that the removal of the projecting tibia with reconstruction of the ankle-joint may be a simpler solution for old uncorrected fractures of this type and would avoid the very extensive operation ordinarily required and which in this case could not be completed because of the circulatory disturbance produced when the soft parts were put under tension.

In reply to questions, Doctor Hitzrot said that there was sufficient cartilage on the upper surface of the astragalus to prevent ankylosis and the best weight-bearing surface of the tibial plafond was the central part. In the reconstruction the astragalus could not be completely brought forward owing to the enlargement of the tibia.

As to astragalectomy, his former experience in this type of fracture had been disappointing. In this case, and in the case of aviators who have crashed, where there are shattering fractures of the lower end of the tibia which also involve the astragalus, an astragalectomy gives a poor weight-bearing foot which is also deformed; also, when astragalectomy is done, the patients walk with considerable more difficulty than that demonstrated by the man shown here.

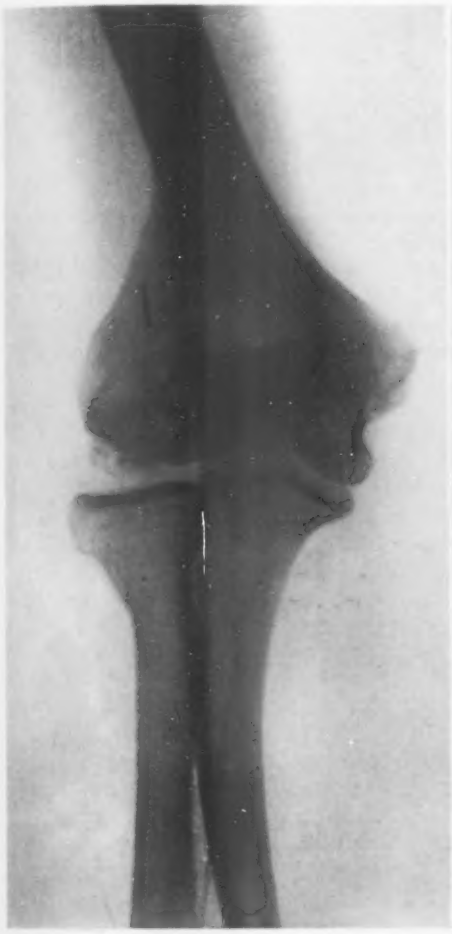

FIG. 4.—Old elbow-joint fracture—anterior view. FIG. 5.—Old elbow-joint fracture—posterior view.

## ELBOW-JOINT FRACTURE WITH UNUSUAL LATE CHANGES

DR. JAMES M. HITZROT presented a lad, aged seventeen, who was referred to him June 6, 1929, with a history of injury to his right elbow nine days before. The elbow was caught in a swinging door and squeezed. Since then his elbow has been swollen, painful and the motion had become markedly restricted. Ten years ago he fell fracturing this elbow. Until the present injury he has had good use of the arm except that he could not bend or straighten it completely.

When seen by the reporter the elbow was swollen; the chief points of tenderness were over the head of the radius, and the posterior aspect of the joint. In the region of the head of the radius there was a large bony mass which seemed to be a large radial head. Flexion at the elbow was limited to about ten degrees beyond a right angle and extension to about forty degrees beyond a right angle. Motions within these limits were quite painful. Pronation and supination of the forearm were both limited but not painful.

X-rays (Figs. 4 and 5) taken June 6, 1929, at the New York Hospital, showed an alteration in the size and shape of the lower end of the right humerus with a change in the shape of the articular surface and widening and distortion of the whole lower end, indicative of an old healed fracture. In addition there was an irregular mass of partially calcified bone or cartilage in the region of the lateral condyle which was irregular in shape, but which could only be seen in the anterior posterior view. The head of the radius was unusually large and the epiphysis had apparently united.

He was admitted to the New York Hospital June 12, 1929, and operated upon on the following day, by an incision over condylar ridge of humerus extending down over the head of the radius. The orbicular ligament was divided and the head of the radius removed through the neck. It was about three times the size of the normal head. Attached to the capitellum was a loose fragment, partially cartilage and partially bone. This was removed and involved the outer half of the capitellum, evidently indicating an old injury to this structure which had not united. During the manipulation of the joint a small loose body about the size of a good-sized pea escaped from the joint. Just where it originated could not be determined, but in all probability it was a fragment from the cartilaginous surface in the region of the fracture of the capitellum. At the completion of the operation and under an anæsthetic it was possible to make practically complete motions of the arm in all directions. He made an uninterrupted convalescence and his motion at the elbow now is practically complete. The case shows a very unusual change in the head of the radius as a late result in an elbow fracture in a child.

## SCLEROSING OSTEOMYELITIS OF CARPAL SCAPHOID

Dr. James M. Hitzrot presented a woman, aged twenty-nine, who was referred to him May 15, 1929 on account of pain in her left wrist. About eighteen months before she had dropped a tire on her left wrist while changing a punctured tire on her car. The wrist at this time became swollen and painful. Although most of the swelling disappeared rapidly, the pain has remained constant. It is dull, boring and aching in character. When she holds anything in her left hand it caves in from lack of strength. For the past three months the pain has begun to affect her whole arm so that she hates to use her left hand for anything. Her past history is negative for previous injury or disease to this wrist. No rheumatism. Scarlet fever, diphtheria, measles, mumps and pneumonia when a child. Family history unimportant.

On examination the left wrist was slightly swollen on the dorsum in the snuff box. Active motions at the wrist were done slowly and were limited. Grip of the hand was weak and when she gripped anything she touched the snuff box as the region of pain. No superficial tender areas nor bone displacement could be found. Forced movements, especially radial adduction gave her pain in the region of the snuff box.

An X-ray report (Fig. 6) showed in the distal half of the left scaphoid

bone a ringlike shadow of sclerosed bone with a darker decalcified appearing central portion. In this darker area is a rounded very dense piece of bone about the size and shape of a green pea. Whether this is Garre's disease or some type of sclerosing bone tumor it is impossible to say. The question of diagnosis was a puzzling one. All clinical blood tests were negative. She was operated upon on May 20, 1929, by a two-inch incision beginning from the styloid process of the radius and extending down to the metacarpal of the thumb between the extensor tendons. This exposed the base of the the scaphoid. The bone at the junction of the capsule and the ligament was distinctly altered in character as compared to the normal bone above it. That is, it was quite white and looked almost like ivory. With a rongeur a piece of this bone was chipped away and it was just as hard as ivory. After the surface had been removed there was a distinctly circumscribed plug of bone which was dense and hard which was separated from the normal bone by a fairly definite capsule of less dense bone.

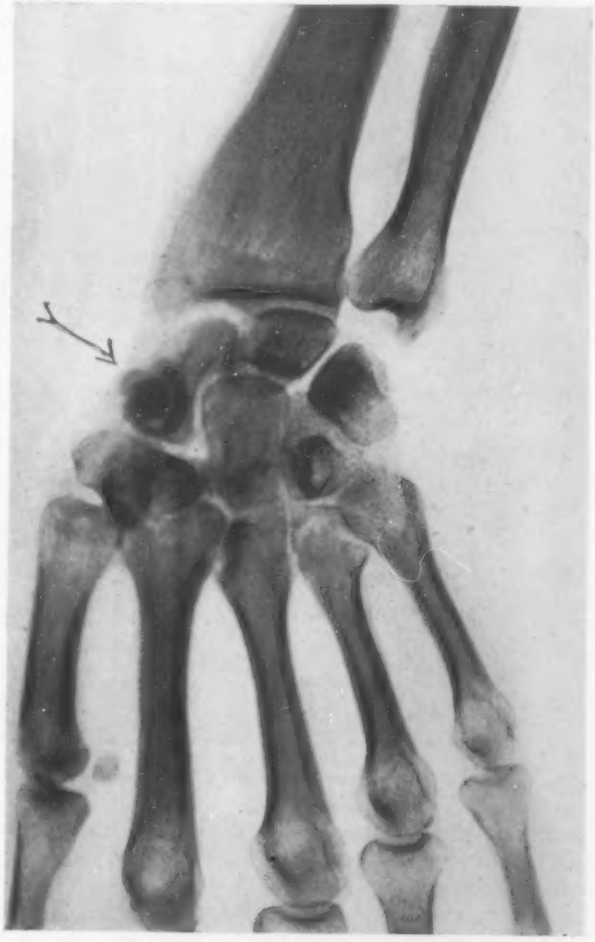

FIG. 6.—Sclerosing osteomyelitis of carpal scaphoid.

The mass was removed in one piece and looked almost like a little plug of ivory inlaid in the bone. The remainder of the bone was apparently normal except that it was a little harder at the margins surrounding this dense bone. This hard bone was then drilled in two places with a small drill, opening into normal vascular bone above. The edges were smoothed off and the soft parts turned into the cavity, leaving the ligamentous attachments from the radius in the cavity.

*Pathological report* by Dr. Elser.—Specimen consists of a small ivory peg removed from the scaphoid bone. It is assumed that this is an instance of Garre disease, although a very unusual location for this type of lesion. The specimen is irregularly spherical in shape measuring between six and seven millimetres in diameter, also several small bone fragments. The peg is very hard to cut.

Histological section of one of the smaller fragments shows fairly normal bone. Sec-

tion of the peg-shaped piece shows very dense atypical bone. The lamellæ are irregular. Lacunæ are present but small and no marrow cavity is found.

The patient made an uneventful recovery. X-ray taken October 22, 1929, shows the process healed with a dense shadow in the involved area. Her pain has gone and the wrist is restored to full use.

## DIVERTICULUM OF ŒSOPHAGUS

DR. ARTHUR S. MCQUILLAN presented a woman, age fifty, who was first seen in September, 1929, complaining of a choky sense in throat and a desire to clear same. Often regurgitation of food, especially liquids, and often a husky voice and sense of oppression in chest. First symptom was noted eight months previous, when she complained of regurgitation of a piece of chicken eaten one week previous. This lodgment of food in the

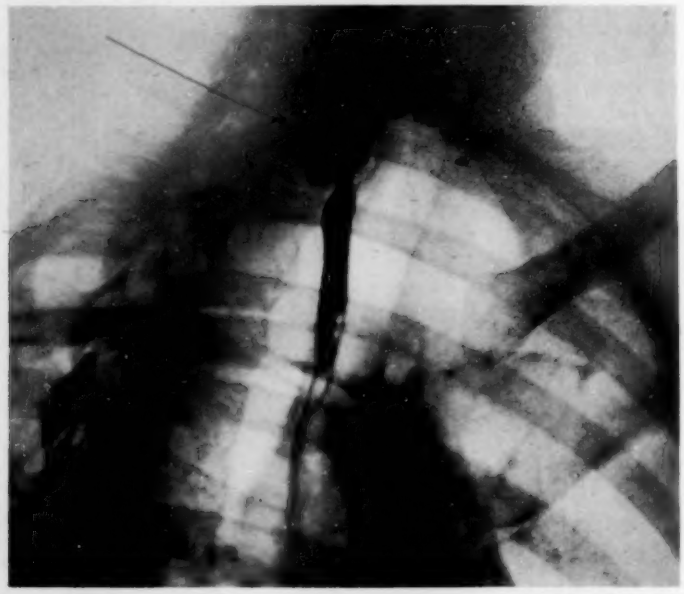

FIG. 7.—Diverticulum of Œsophagus. X-ray immediately following barium meal.

neck was the most annoying symptom and often the patient would press on left side of neck (just to left of trachea) to relieve this disagreeable symptom. Pressure in this area would cause a gurgling sound (as if air and fluid were escaping) and this could always be elicited by patient and examiner. Saliva could usually be made to regurgitate on pressure over this area.

Physical examination was negative with exception of pressure signs in left midpart of neck between trachea and sterno-mastoid muscle. X-ray immediately and one-half hour after swallowing a barium meal showed a rounded pouch of œsophagus situated on the posterior aspect towards left side; at level of second dorsal vertebræ. Outline smooth and regular. Inlet could not be visualized. No obstruction and no other deformities.

The woman entered hospital September 27, and was operated upon September 28, 1929, eight months after noting first symptom. The incision was along the anterior border of the sterno-mastoid muscle down through deep fascia overlying prethyroid muscles. The omohyoid had to be severed to obtain good exposure. By retraction of sterno-mastoid and carotid sheath

contents laterally, the thyroid gland and trachea medially, the space lateral to trachea and œsophagus was exposed. The œsophagus and diverticulum were easily identified, the diverticulum being situated just lateral to œsophagus and to the posterior half of trachea with fundus pointing down towards mediastinum. By careful dissection and traction on diverticulum, which was bound in position by loose areolar tissue, the pouch was easily freed and its neck, one-third the diameter of the fundus, was found to come off the œsophagus on its posterior aspect. The diverticulum was pulled into wound and its neck ligated near œsophageal wall with Pagenstecher, the ends left long so as to reach through wound in neck of patient. Another similar suture was attached to fundus of diverticulum and the ends left long for traction purposes. The diverticulum was then surrounded with rubber dam, and with a cigaretted drain on upper and lower sides, the wound in neck was

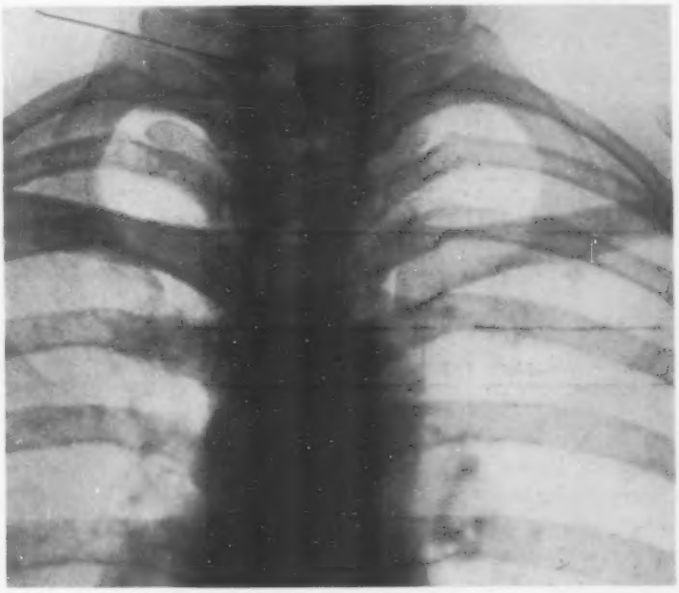

FIG. 8.—Diverticulum of Œsophagus. One-half hour after barium meal.

closed to drains. The whole procedure was simple and there was surprisingly little demand for securing hæmostasis. The time consumed was forty-five minutes and patient was in excellent condition at end of procedure. The most important structure to be kept in mind is the inferior laryngeal nerve which was protected to some extent by the thyroid gland.

*Post-operative Record.—First day.*—General condition was excellent with only slight sero-sanguinous discharge.

*Tenth day.*—The diverticulum on slight traction came away in one necrotic mass. There had been an offensive odor on one or two occasions. The patient was able to swallow liquid and solid food from the first.

*Tenth to Eighteenth day.*—The sinus gradually narrowed down. There was a moderate amount of leakage of liquid food which soon diminished in amount and completely stopped during the fourth week post-operative. At present time (tenth post-operative week) the sinus is practically closed, there being left an area of granulation tissue one-quarter inch in diameter.

DR. EDWARD W. PETERSON referred to his report, some years ago, of five cases of diverticulum of the œsophagus operated upon by the one-stage

method. One death in this series had convinced him that the two-stage operation was much the safer and wiser procedure.

Dr. Charles Gordon Heyd stated that the consensus of opinion in regard to the operative treatment of diverticulum of the œsophagus was that the two-stage operation was the procedure of choice. The outstanding danger in manipulation of œsophageal diverticula was the development of infective mediastinitis. This complication could ordinarily be prevented with the two-stage operation. At the first stage identification of the diverticulum and its marsupialization allowed closure of the superior thoracic aperture and ten days later the diverticulum could be excised under local anæsthesia and the neck inverted. Doctor Heyd could not quite see the reason that Doctor McQuillan had placed a Pagenstecher around the neck of the sac. After this was done gangrene of the sac was an inevitable result and of necessity the wound in the neck must heal by granulation. If œsophageal leakage does occur after the second operation healing can be expedited by the introduction of a Levine tube through the mouth and nose and by means of the tube feedings may be maintained without leakage through the œsophageal fistula. In the second stage operation the recovery should be prompt and ordinarily recovery by granulation should not occur. Doctor Heyd showed some lantern slides demonstrating the technic of the two-stage operation.

Dr. John Douglas also endorsed the two-stage operation. He showed some X-ray films of and a specimen from a woman he had operated on who for twenty years had shown all the symptoms Doctor McQuillan had described. Doctor Douglas saw her in May, 1928, but at that time she refused operation. Subsequently, her symptoms becoming more pronounced, she finally consented to operation which was done last June. The method of operation was that advocated by Doctor Heyd and, under local anæsthesia, the sac was freed very easily. Eleven days after the primary operation (reports from the Mayo Clinic recommend eleven to twelve days' interval) the sac was separated and excised, using only morphine and hyoscin. Novocaine was not necessary as the patient slept through the procedure. There were one or two points of interest, the first being in regard to Doctor McQuillan's statement that the entrance to the sac was posteriorly. It is always posteriorly in these cases in which the diverticulum occurs in the neck. The radiographer's report in the case here reported shows the entrance to the sac to be on the left side, but they always originate posteriorly. Another point of interest is the fact that in looking at this specimen one sees a very thick wall. One method of operation consists of invaginating the sac; if most of the cases had a wall as thick as this one no infolding could be done. There was another point of interest: This operation was done only six months ago, and the patient came to see Doctor Douglas recently in routine follow-up, although there was no return of symptoms, and he was able to pass a large bougie through the œsophagus to the stomach with no difficulty; but to prove she was well he had a röntgenogram taken which showed the

presence of a small pouch. So there is no certainty of complete cure in these cases and it would be interesting to see some röntgenograms of Doctor McQuillan's patient six months from now.

Doctor McQuillan, in closing the discussion, denied that he had meant to claim that his method had any great advantages over the usual two-stage operation, but he claimed that it is true that the wound heals by secondary union in this method. Healing *per primam* in the marsupialization method does not always occur as there is danger of infection in the second stage; also, two major operations are necessary to accomplish the same result. Doctor McQuillan agreed with Doctor Peterson, Doctor Douglas and Doctor Heyd that the one-stage operation for œsophageal diverticulum is too risky to be considered a safe procedure for if infection into the mediastinum occurs it is fatal. In answer to Doctor Heyd, the Pagenstecher was placed on the neck of the diverticulum close to the œsophagus in order to cut off circulation, so that the diverticulum would slough off at the end of ten days or two weeks. It is probable that at the end of two or three days enough adhesions are formed mechanically by the drains to wall off any later infection. The usual thickness of the diverticulum wall delays the necrosis. During the first ten days the discharge, only slight in amount, was of a serious nature and it was only after the sac had sloughed that there was œsophageal leakage for a short time.

## GOITRE WITH ACUTE COMPRESSION OF TRACHEA

Dr. Arthur G. McQuillan presented a girl whom he had first seen in April, 1927, when she was ten years of age. She was found to be in an extreme degree of dyspnœa and collapse. The thyroid gland was enlarged, one firm nodule in isthmus, one small one in the left lobe and another twice the size in the right lobe. Respiration was extremely labored and of the inspiratory type (expiration free and inspiration difficult). There was apparent obstruction to inspiration. Breathing was possible only in the sitting posture. She kept her head extended with chin elevated, giving the appearance of a stiff neck. She was afraid to recline or relax as in the reclining posture respiration became more embarrassed. She had had no sleep for a week except at intervals. There had been dysphagia for three weeks. The result was exhaustion and a loss of fifteen pounds weight. So great was the inspiratory pull that the lower chest flared with a groove three inches in depth, the latter noted by the parents only since the difficult breathing. There was acetone breath, marked cyanosis of face and finger tips, pulse 120, respiration 24 and temperature 100° F.

The child was said to have had a goitre for four years, noted in right side of neck. Two years later a similar nodule appeared in left side and a central swelling appeared during the past year. Trouble began in January, 1927, she began to have occasional obstructed breathing only on exertion and for the last six weeks continued obstructed breathing and dysphagia. There was a history of iodine medication with no results. The patient was born and bred in New York City, and the father was said to have had a goitre at the age of six and that it disappeared in the course of a year.

The child was taken to the hospital immediately and operated with only one object, to do a decompression and relieve respiratory obstruction. In the sitting posture and with novocaine infiltration anæsthesia, the thyroid

gland was exposed, showing a three-inch cystic nodule of the isthmus. While attempting to dislocate this, the patient fell back in syncope. In a hurried procedure, this cyst with four others surrounding the trachea was ruptured and respiration became easy. As nearly as one could tell, there was a systic nodule of the isthmus, two multilocular ones in the right lateral lobe, and posterior aspect of trachea, and two smaller nodules on the left lateral aspect. All were tense, wedging the trachea centrally and filled with fluid contents and degenerated soft thyroid tissue. There was a considerable amount of thyroid tissue posterior to the trachea. Thus the trachea was in the center of these cystic thyroid masses, which in turn were partly wedged into the space of the superior thoracic aperture. As inspiration would pull the trachea down, the mass of thyroid tissue completely surrounding it was wedged between the bony margin of the superior strait and the centrally placed trachea, resulting in partial collapse of the trachea and obstruction to respiration. Hence the effort on the part of the patient to keep her neck extended and chin elevated. On account of the grave condition of the patient, only a moderate amount of thyroid tissue was excised.

The patient made a good recovery and went along for about five months, when slight obstruction to inspiratory breathing was noted especially on exertion. This increased fairly rapidly. In January, 1928, (eight months following the first procedure), operation was attempted. Under ether anæsthesia with a No. 17 silk catheter in the trachea, with a funnel covered with gauze on proximal end of catheter, to facilitate the ether anæsthesia. The patient did poorly, so that at the end of thirty-five minutes' procedure, with considerable cyanosis and poor quality of circulation, it was decided to stop, with the result that only a three- by two-inch cyst, involving isthmus and right lobe of thyroid, was excised. There was some question as to whether or not the catheter went through the narrowed part of trachea. However the condition of the patient did not warrant further attempt of introducing the catheter. Following this operation the breathing became normal again and the patient made a good recovery.

Again she went on for a six-month period with only slight respiratory embarrassment on exertion, but from then on difficult breathing grew steadily worse, until in September, 1929 (nine months following the second operation), it became necessary to operate again to relieve the obstruction to breathing, which was becoming serious. This time a catheter was successfully passed through the tracheal narrowing. The thyroid tissue was so badly scarred and adherent to the trachea, that a subtotal thyroidectomy had to be done, leaving only about one-quarter of the right lobe in region of upper pole. The trachea was left free of thyroid tissue in its whole extent and it showed lateral flattening due to long continued pressure. Pallor and puffy eyelids, noted for several weeks following operation, have entirely cleared up at present time on thyroid feeding.

## A CLINICAL STUDY OF CALCIFIED NODES IN THE MESENTERY

Dr. Hugh Auchincloss read a paper with the above title for which see page 401.

Dr. Allen O. Whipple expressed the appreciation of the surgeons at the Presbyterian Hospital for this work that Doctor Auchincloss has originated. It had made them all believe that the diagnosis of calcified mesenteric lymph nodes should be differentiated from the lesions of the appendix. Doctor Whipple's own experience in these cases, including some of those

whose X-rays were reported by Doctor Golden, had been limited to eight cases, all in young women under thirty years of age and three in physicians' daughters. Two of the cases were observed for a period of five years before operation and showed progressive increase in the size of the deposits of calcium in the ileocæcal mesentery. Three of the patients were seen after a diagnosis of acute appendicitis had been made and the three were operated on later after the diagnosis of calcified mesenteric lymph nodes had been established. The other five cases were diagnosed as chronic appendicitis but

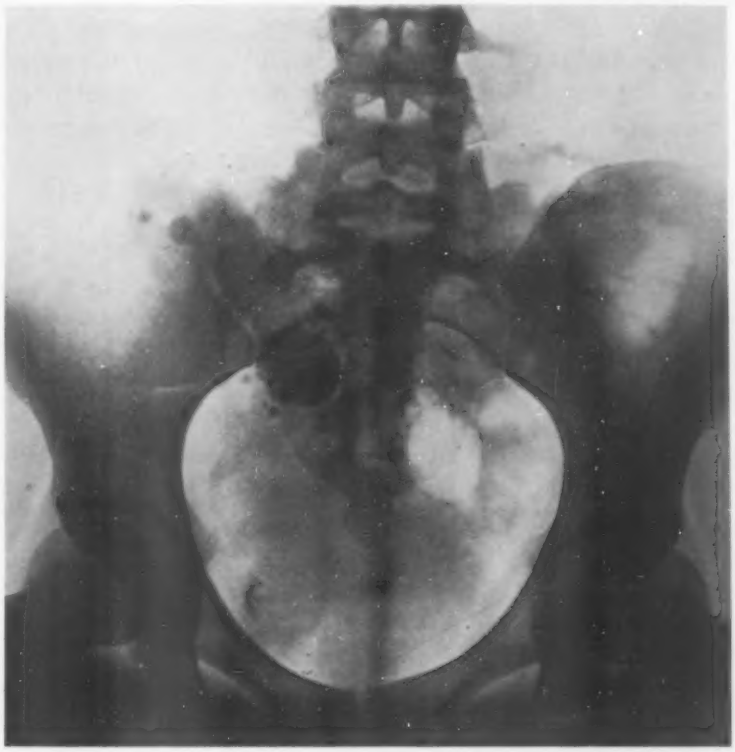

FIG. 9.—Multiple small and large, completely and incompletely calcified glands, overlying the right half of the pelvis. The large gland is still soft, with omentum adherent over it, and its contents are incompletely calcified.

later on the X-rays showed them to have chronic lymphadenitis. Two of these cases operated upon showed typical tubercles in sections under the microscope and this was confirmed when guinea pigs were inoculated. Doctor Whipple considered it striking that all these eight cases were in young women under thirty years of age.

DR. JOHN F. CONNORS said that fifteen years ago he operated on a child twelve years of age for appendicitis. She had pain in the right iliac fossa. At operation a normal appendix was found but in the ileocæcal region there were calcified lymph glands. Doctor Connors did not know just what they were but he felt they indicated a bad prognosis, so he sewed up the wound and told the father he was afraid she was doomed. He did not know what

the outcome would be but advised taking her to the country to live. This was done and the girl has remained healthy ever since. Two years after this experience Doctor Connors was called to see a doctor's daughter for appendicitis; at operation he found the same condition of tuberculous mesenteric glands. He gave the same advice as in the first case and this patient too has remained well.

Dr. John Douglas asked if Doctor Auchincloss had any figures showing the percentage of occurrence of this lesion without causing symptoms. The speaker has had a few cases in which he has found enlarged lymph nodes and calcified lymph nodes in the ileocæcal region and in the mesentery of the small intestine and in one case he found some in the region of the duodenum. He was under the impression that in a number of these cases where shadows are found in the X-ray plates where examination has been made for gall-stones, or kidney-stones, the shadows are in reality due to calcified glands, particularly in those cases where it is very difficult to identify those shadows with the condition for which examination is being made. They are not always in the ileocæcal region. Doctor Douglas asked if Doctor Auchincloss could tell how many patients with positive X-ray findings are symptom-free, or in whom the symptoms cannot be linked up with the presence of shadows indicating the presence of calcareous glands.

Dr. Edwin Beer showed some pictures of a case similar to some of those of Doctor Auchincloss. At operation one of the glands as large as a walnut was still in a soft condition, full of calcareous material and there were omental adhesions attached to the gland which had perforated the mesentery. The material was full of lime salts and pus. There were no tubercle bacilli. Numerous other glands were solid and calcareous. In genito-urinary X-ray work the speaker sees a very large number of these shadows, some of which cause confusion, especially when renal colics are present. He had the impression that calcified glands never are associated with symptoms when they are hard and small in size, whereas the large, soft ones are apt to give more definite symptoms, due to adhesions, perhaps simulating appendicitis or adhesions or even ileus.

Dr. Ross Golden (by invitation) said that in going over the literature a year or more ago he discovered some interesting information bearing on the question of the mechanism of infection of tuberculous mesenteric lymph nodes. Orth, the German pathologist, did some feeding experiments with calves before the identification of the tubercle bacillus which led him to the conclusion that the "poison" of tuberculosis was of the type that could pass through the intact intestinal wall. In 1890, Dobroklonsky, a Russian working in Paris, fed guinea pigs with a gycerine culture of tubercle bacillus. He made sections at varying intervals after the feeding. He found that the organisms very soon were present in the mesenteric lymph nodes, and proceeded to produce a typical tuberculous process. Some of his sections showed tubercle bacilli lying between the epithelial cells of the lining of the intestines and organisms were seen projecting from the intestinal walls into the lumen.

Other pathological and experimental evidence is present in the literature tending to confirm this same assumption; that is, that tubercle bacilli can produce a disease of the mesenteric lymph nodes without leaving any trace of their effect upon the intestinal wall.

X-ray examination was first recommended by Cornor, an English surgeon. In 1905 he reported a case of abdominal pain in a man of approximately middle age with a dense rounded shadow in the röntgenogram exactly overlying the right kidney. This was thought to be a kidney-stone. However, for some reason the pain was thought to be of nervous origin and the patient was discharged. The continuation of the pain, however, led to an exploratory operation at which a caseo-calcareous node about the size of a walnut was found exactly overlying the right kidney. It was curetted out and the patient had no more pain. In his second communication on this subject in 1909 Cornor again recommends X-ray examination as an aid to the diagnosis of tuberculous lymphadenitis. It is also mentioned by two or three German writers at this period. Apparently it is not mentioned again in the literature until 1926 when an article by Dunham and Smythe appeared. Doctor Auchincloss has described very nicely the X-ray appearances of these shadows. They may be very difficult to see if they overlie the spine. They may overlie the spine at one examination and not at another. If a patient breathes during the exposure the motion of the node may cause its flaky density to be blurred, giving it the appearance of a homogeneous dense shadow more like a kidney-stone. In one case such a blurred node shadow was misinterpreted as a kidney-stone and the patient submitted to an unnecessary operation. A film taken after the operation showed the shadow without motion and it was a typical calcified node. Node shadows have been seen apparently blurred by motion even though the patient apparently held his breath perfectly well. The only obvious explanation seems to be that the node lay near enough in the mesentery to the margin of the intestine to be shifted in position by peristaltic movements during the time of exposure.

In regard to the differentiation of so-called chronic appendicitis from the disease under consideration, the barium meal may give some helpful information. If the appendix is filled one can sometimes demonstrate that the tender point lies not over the appendix but medial to it or slightly above it. In some cases it has been possible to demonstrate that the tender point exactly coincided with the shadow of the calcified nodes.

Doctor Auchincloss, in closing the discussion, replied to Doctor Douglas with regards to the prevalence of these glands without symptoms, that this undoubtedly did occur in many instances; and in many other instances, though symptoms actually did exist, they were too mild to make the patient go to a doctor. The records in the British army showed that in a presumably healthy group of men calcified glands were found in about 25 per cent. These men presumably had high resistance and showed well walled off lesions. The statistics of Doctors Dunham and Smythe give a fair indication of their prevalence in children. Doctor Auchincloss stated that he did

not wish to give the impression that when calcified nodes were giving symptoms that the symptoms were necessarily due to the nodes, for several cases of acute appendicitis verified at operation have had calcified nodes. On the other hand, one finds great interest in those cases that present symptoms after the appendix has been removed, or some other operation done. It is very difficult to accurately estimate the proportion of cases where the symptoms are due to the calcareous nodes alone, but there was no doubt that such cases did exist. Doctor Beer's case showed a very typical picture with extensive involvement. Doctor Auchincloss was in doubt—how much more frequently large soft nodes gave symptoms, as Doctor Beer stated, than when they were small and hard. He did not believe the size of the nodes was the only criterion. In fact the patient presented before the Society this evening showed several small nodes that were pretty hard and had become calcified within four years' time. The little nodes may give symptoms as well as the large ones.

### STATED MEETING HELD JANUARY 8, 1930

## POLYPOSIS OF CÆCUM

Dr. J. William Hinton presented a young girl seventeen years of age, who was admitted to Bellevue Hospital, Fourth Surgical Division, October 9, 1929, on account of abdominal pain, loss of weight, and constipation alternating with diarrhœa. She had been operated upon in August, 1928, for chronic appendicitis in another hospital. She was somewhat improved by the operation but has never been entirely well. A complete description of the operative findings could not be obtained from the hospital but a verbal report from the operating surgeon was to the effect that the patient had ulcers of the intestine and chronic appendicitis, an appendectomy being done and no attempt being made to remove the ulcers of the intestine.

The patient stated that in April, 1929, she began to lose weight, had pain in epigastrium and constipation. She was put on a milk diet without any improvement and had various diets given for the next few months. In August, 1929, she had a gastro-intestinal X-ray series which was pronounced negative, although she continued to lose weight until four weeks before admission when she had an attack of diarrhœa which lasted for two weeks. This was followed by constipation. There was no history of cough, or night sweats, but she had lost thirty-five pounds in weight. She ran a septic temperature ranging from 100° to 103° while in the hospital. Chest examination was negative with negative X-rays. There was a mass in the right lower quadrant which was tender to pressure. Blood counts were normal. Leucocyte count 6,400 to 6,800, with polymorphonuclears ranging from 70 to 75 per cent. Wassermann negative. A gastro-intestinal X-ray series revealed the stomach and duodenum normal. Barium enema revealed a deformity of the ileum, cæcum and ascending colon. On October 22 she was transfused with 500 cubic centimetres of blood. October 25, 1929, under gas, oxygen, ether anæsthesia, the abdomen was opened through a right rectus muscle splitting incision. On opening peritoneum there was found a large mass which was the size of one's fist, in the right lower quadrant, the mass being attached to the parietal peritoneum in the region of the brim of pelvis. The mass was freed from the parietal peritoneum with only slight difficulty, and the cæcum, ascending colon and ileum easily delivered. The

mass gave the appearance of a tubercular infection of the cæcum which involved the lower ileum and ascending colon. There was no other evidence of involvement of the gastro-intestinal tract, and the peritoneum was normal. There were some enlarged mesenteric lymph glands. A typical Friedrich's operation was done, removing the lower six inches of the ileum, cæcum and ascending colon; and one-third of the transverse colon. The transverse colon was closed and then an end-to-side anastomosis was done, using No. 1 chromic gut for the anastomosis, taking three rows of sutures. Abdomen closed in anatomical layers, with small rubber tissue drain in the right lower quadrant.

*Post-operative course.*—The patient's convalescence was uninterrupted for the first five days, then she developed a fæcal discharge from the wound and this continued for four weeks. Her appetite returned within a few days following operation. She had a normal bowel movement daily and her temperature was normal after the first four weeks. She was discharged from the hospital December 6, with a granulating wound which was slightly infected.

*Pathological Report.*—(Doctor Symmers.) Macroscopic Examination: The specimen consists of the terminal 12 centimetres of ileum and the proximal 12 centimetres of cæcum, in one piece. The peritoneum covering the external surface of the gut is entirely normal. The mucous surface, on the other hand, shows two longitudinal ulcers, each measuring about 4 by 2 centimetres. The base of the ulcer is grayish red in color and finely granular. The mucosa at the edges of these ulcers is redundant and thrown into thick, bluish red, velvety folds and mound-like or actual polypoid masses. Of the latter, one is nearly 2½ centimetres in diameter and partially obstructs the lumen of the gut, Microscopic examination of the ulcerated areas shows the presence of simple ulcers, the bases of which are fairly rich in granulation tissue while the mound-like and polyphoid masses at the periphery of the ulcers are found to be made up of hyperplastic mucous glands. These mound-like and polypoid masses obviously represent an attempt on the part of the mucous membrane to regenerate itself in order to compensate for loss of substance occasioned by the process of ulceration. The lesion throughout is a simple inflammatory and regenerative one and shows no histological relationship to any of the granulomatous lesions, including tuberculosis.

*Pathological diagnosis.*—Mucous polyposis.

## CHOLEDOCHOGASTROSTOMY FOR SCAR TISSUE OBSTRUCTION OF THE COMMON DUCT

Dr. J. William Hinton presented a woman, forty-eight years of age, who was admitted to Post-Graduate Hospital June 25, 1929, on account of jaundice, itching of skin and pain in epigastrium for the past few weeks with increasing jaundice and itching. She had been operated on for an abdominal tumor in 1919, for a goitre in 1925, and a gall-bladder in October, 1928. She dates her present complaints back about four weeks when she noticed her skin getting yellow and she was bothered by some discomfort in epigastrium. No severe pain but an uncomfortable sensation in the upper abdomen, accompanied by belching of gas, without vomiting. Her condition gradually became worse and ten days before admission she was bothered with itching of the entire body, as well as increasing jaundice. In October, 1928, she was operated upon for a gall-bladder condition, after which she drained bile until the early part of January, 1929. Then the wound healed and she remained in fairly good health until one month ago when she began having epigastric discomfort and jaundice.

Report from previous operation at the St. Augustana Hospital, Chicago, Ill.—Stomach and duodenum normal. Gall-bladder green in color and

thickened. Enlarged glands along the duct, no stones palpated. Appendix, adhesions around the cæcum, subacutely inflamed. Uterus, normal in size and position. Adnexa in adhesions. Operation, cholecystectomy and appendectomy.

When admitted to the Post-Graduate Hospital she was thin and undernourished, with skin and sclera markedly jaundiced. She was twenty pounds underweight. Her neck reveals a scar from goitre operation but no evidence of thyroid enlargement at present. Abdomen, upper right rectus scar, firmly healed and also a lower midline incision firmly healed. Due to scratching from itching, she had numerous abrasions over arms, legs and body, with pruritus ani. Urinalysis negative. Bleeding time four minutes. Clotting time began five and one-half minutes, completed six minutes. White blood count and differential count normal. She was given daily hypodermoclysis 1000 cubic centimetres of 3 per cent. glucose, and 15½ grains of calcium chloride, intravenously, for four days.

July 3, 1929, her abdomen was opened through a right rectus muscle splitting incision, previous scar being excised. On opening the peritoneum, there were moderate adhesions in the right upper quadrant in the region of the gall-bladder bed, but the gall-bladder bed could be exposed without much difficulty, and the stomach was freely movable with duodenum adherent and definitely fixed. On exposing the common duct there was found a definite scar tissue obstruction one inch below the hepatics with a dilatation of the upper inch of the common duct, and both the hepatics. The common duct was opened and both right and left hepatics were probed and found to be patent. The common duct was definitely obstructed from scar tissue. The stomach could be easily approximated to the dilated portion of the common duct. It was impossible to approximate the duodenum to the common duct. A direct anastomosis between the common duct and stomach was done, the steps of which were identical to the steps of a gastro-enterostomy. After completing the anastomosis, three mattress sutures were taken from the stomach to the under surface of the liver to relieve any tension on the suture line between the common duct and stomach. A small rubber tissue drain was inserted through the peritoneum. Abdomen closed in anatomical layers.

The patient's convalescence was uneventful for the first few days. There was no leakage of bile around the drain until the fourth day when the patient saturated the dressing with bile. On the fifth day there was only very slight leakage of bile on the dressing, and on the sixth day it entirely disappeared. Cigarette drain removed on the sixth day, and from then on there was no leakage of bile. At this time it was noticed that the jaundice was definitely subsiding and the itching improving. The patient continued to improve and was discharged from the hospital on the twentieth day, at which time she was practically free from jaundice, and itching had entirely disappeared. The wound had healed with the exception of a slight area at the lower angle which was infected.

She was last seen November 11, 1929, at which time she felt perfectly well and was free from jaundice. She had gained eighteen pounds in weight since the operation.

The one reason for doing this operation was the ease with which the stomach could be approximated to the common duct for the anastomosis without tension on the suture line.

Dr. Seward Erdman noted that Doctor Hinton in this case had been able to achieve the ideal condition of approximating a mucous lined surface to a mucous lined surface in repairing an obstructed bile duct. Considering

his own cases and those he had seen operated upon by others in the New York Hospital, he regarded it as unusual to be able to secure such exposure of the dilated portion of the duct that one could approximate it to the stomach. Other methods have been tried and have seldom proved satisfactory for any length of time, when dependent upon a tube of fibrous tissue. Often the case will do well for six or eight months, but it tends to contract down and then something further has to be done. Doctor Erdman considered that Doctor Hinton's ability to get a continuous mucous surface in this case should maintain the patient in perfect health indefinitely, as far as the common duct obstruction was concerned, and he believed that a report on this case in a year or two would prove this to be true.

Dr. John Douglas referred to three cases of his own of post-operative stricture of the common bile duct. He had reported two of these cases in the Annals of Surgery in 1926. One of these patients called on him three weeks ago, suffering from rheumatism, and he learned that she had recurring attacks of mild jaundice, with slight rise of temperature, for two or three years after operation, but for the last five years she has been symptom free. The second case had recurring attacks for a few years after operation but has been symptom free for three or four years. Apparently the cause of post-operative symptoms after reconstruction operations in these cases was a residual narrowing of the duct because whenever they had recurrent attacks, a proprietary preparation of bile salts had tided them over the attacks of cholangitis by increasing the flow of bile, thinning it down and clearing out the infection. The third case referred to, which has not been reported, was a woman who had undergone a cholecystectomy at which time there had been perforation of the gall-bladder with abscess outside the gall-bladder from infection. The common duct was drained for some time and then healed up. A few months afterward she developed an obstructive jaundice. Doctor Douglas operated on her about three years ago with the expectation of finding dense adhesions but in fact found very few. It was very easy to find the dilated common duct. The obstruction in the common duct was low, lower than where the cystic duct comes off, for there was room to do a lateral anastomosis between the common duct and the duodenum which came in apposition very easily and the anastomosis never leaked a drop. She has remained well to the present time. When the operation can be done in this way the results are the most favorable, but in most of the cases the obstruction is very high up and at the junction of the hepatic with the common duct where such an operation cannot be done, and some form of tube anastomosis gives the best results.

Doctor Hinton, in closing the discussion, said that he also believed that it was only in a few cases that this operation could be done. The reason he undertook it was because as conditions presented themselves in the abdomen, the operation seemed easy and simple. In reviewing the literature he had seen reports of cases in which the left hepatic duct had been anastomosed to the stomach when the obstruction was high up in the common duct.

## SEVERE GASTRIC HÆMORRHAGE TWO YEARS AFTER PARTIAL GASTRECTOMY FOR PYLORIC ULCER

DR. J. WILLIAM HINTON presented a man, thirty-one years of age, who was admitted to Bellevue Hospital, Fourth Surgical Division, December 1, 1929, on account of bleeding from rectum and vomiting of blood. The man began having pain after meals which would be relieved by taking food, in 1918. He was under the care of different physicians at that time and was placed on a diet. In 1923, not having been relieved of his pain, he entered Bellevue and had X-rays taken which revealed a duodenal ulcer. He was treated on a Sippy diet for two weeks at that time. He followed this diet with some regularity after that but continued to have pain.

He was admitted to the Fourth Surgical Division December 17, 1925, complaining of passing tarry stools for twenty-four hours, and bright red blood a few hours before admission. This was the first evidence of any hæmorrhage from the gastro-intestinal tract, although he had been home on a Sippy diet for two weeks before this admission. Also vomited blood after admission to hospital. Transfused the same day, with 700 cubic centimetres of blood. His abdomen was opened December 28, 1925. There was found on the anterior surface of the first portion of the duodenum, just beyond the pylorus, a crater-like ulcer about 1½ centimetres in diameter. A Finney pyloroplasty was done, using No. 1 chromic for the repair. He was discharged from the hospital January 1, 1926.

In April, 1926, he began to complain of pain in epigastrium, with a burning sensation. Had also lost six or seven pounds in weight. He had followed a Sippy diet regularly since his operation. X-ray examination April 22, 1926, revealed a small niche formation present at the site of the operative procedure, which suggested the presence of an ulcer in that region. He failed to return thereafter to the follow-up clinic.

January 8, 1928, he entered the Post-Graduate Hospital stating that he had continued to have pain since his operation. On January 9, he was operated upon and found to have a pyloric ulcer and a chronic cholecystitis. Partial gastrectomy was done, removing 13 centimetres of stomach, measured on the greater curvature. There was a crater-like ulcer of the pylorus 1½ centimetres in depth, with a lumen 5 millimetres in width. Cholecystectomy was also done. Pathological report revealed a chronic cholecystitis. He was discharged from the hospital January 24, 1928.

He now states that since this operation in January, 1928, he had been perfectly well, until a few hours before admission. On returning home from work he felt weak and dizzy and passed a tarry stool which was followed by red blood. He fainted and an ambulance was called, but before being brought to the hospital he vomited blood. He was transfused December 4, 1929, 500 cubic centimetres of blood being given. He continued to vomit blood for four days after admission. Transfused December 9, with 500 cubic centimetres of blood, after which he passed small quantities of blood in stools until December 18. Discharged from the hospital December 24, and is now under observation in the Gastro-Enterological Clinic.

## ECHINOCOCCUS CYST OF LIVER THIRTY-THREE MONTHS POST-OPERATIVE

DR. J. WILLIAM HINTON presented a woman, twenty-seven years of age, who was admitted to Fourth Surgical Division Bellevue Hospital, March 8, 1927, on account of attacks of pain in right upper quadrant of abdomen with jaundice for the past eight years. According to her history about eight years ago she had an attack of pain in the right upper quadrant which radi-

ated to the back and right shoulder. This pain was very severe. She has had several attacks a year since the first attack and has been jaundiced on a number of occasions. Between attacks she is bothered with belching of gas, sour eructation and heartburn. She states that during the attacks she has been jaundiced, her stools have been clay color. The last attack began eight days before admission when she had a sudden attack of pain in right upper quadrant which confined her to bed and she became jaundiced. This lasted only three or four days and the symptoms gradually subsided until the time of admission.

She was a well developed, nourished woman. Chest negative. Heart regular. The liver edge could be palpated just below the costal margin. There was a mass palpable in the right upper quadrant which was acutely tender with definite muscle rigidity on the right side. No other mass palpable in abdomen, no tenderness in right lower quadrant. No kidney tenderness. Temperature normal on admission.

March 9, 1927, her abdomen was opened through a right rectus muscle splitting incision. On opening the peritoneum a cystic mass, which was approximately the size of a large grapefruit, or even larger, was encountered which was adherent to the parietal peritoneum of the anterior abdominal wall. The mass was dissected free from the peritoneum, and it was found to involve the liver substance. On exploring the abdominal cavity the duodenum was found densely adherent to the mass which was dissected free. Only about one-quarter of the total circumference of the cyst was imbedded in the liver. The gall-bladder was found attached to the cyst but there was no evidence of stones in the gall-bladder and it was apparently normal. The cyst could be dissected out of the liver substance with very little difficulty and after doing so the cystic duct was easily exposed, clamped and divided, and the gall-bladder removed still remaining attached to the cyst. The cyst was not ruptured in removing same. The common duct did not reveal any evidence of stones. The hæmorrhage from the liver tissue was controlled by hot packs and the liver bed packed with iodoform gauze. No other cysts could be felt in either the right or left lobes of the liver.

Her convalescence was quite stormy for the first ten days, due to the destruction of liver tissue in removing the cyst. She began draining bile two days following operation and continued to do so for the next three weeks. On the fourteenth day post-operative, she developed an effusion in right chest which had to be aspirated on three occasions. Cultures were negative. The wound became infected and she had a partial separation of the wound ten days after operation. This gradually cleared up and the patient was discharged from the hospital June 13, at which time the right chest was entirely clear, and the wound was practically healed, and the patient had no complaints. Since leaving the hospital she has been seen in the follow-up clinic at regular intervals, the last visit being October 7, 1928, at which time she was perfectly well and had been since her operation. She was seven months pregnant at the time.

## TREATMENT OF EMPYEMA AND LUNG ABSCESS

Dr. John F. Connors presented three cases, two of empyema and one of lung abscess, illustrating the results obtained in sixteen cases so far treated by a method which he subsequently described.

Case I.—A boy, fourteen years of age, was admitted to Harlem Hospital, November 28, 1929, with a diagnosis of pneumonia. On December 15, 1929, the chest was tapped and pus removed. Culture showed type 1 pneumococcus. On December 17 he was transferred to the surgical service.

On December 18 he was operated upon by the method to be described. In this case the time of dressings was changed to one every day for six days in an attempt to clean the cavity more quickly but it made no difference in the amount of the discharge, and for this reason the number of dressings was greater, this being the thirteenth, and there is now a very small amount of discharge.

CASE II.—Was operated upon November 11, 1929, and was discharged from the hospital December 6, 1929. The operation for empyema was performed following the method to be described. At the time of discharge the wound was entirely healed and has remained so since.

CASE III.—This patient was admitted to the medical service with a diagnosis of lung abscess. He was transferred to the surgical service and operation was performed August 12, 1929. When the parietal pleura was opened there was no collapse of the lung. After three punctures with a needle the pus was withdrawn from the left lower lobe about one and one-half inches from the surface. With the finger the normal lung tissue was penetrated into the abscessed cavity. The cavity was then packed tightly with washed iodoform gauze and the patient was returned to bed. Within forty-eight hours the foul smelling expectoration had almost stopped. This patient was packed twelve times and was discharged from the hospital thirty-one days after operation. The noteworthy factor of this case was that during the sixth dressing, on August 23, in removing the gauze the patient coughed and expelled not only the gauze but what appeared to be a cast of the abscess. From then on the wound remained very clean.

Doctor Connors said that in presenting these cases he was submitting for consideration the method of treatment and management of empyema cases as used by him at Harlem Hospital. Sixteen cases had been thus treated, but while the number is admittedly small, he had been sufficiently encouraged by the results to submit it to this society for discussion, criticism and suggestions for improvement.

Briefly the method is as follows: One hour before operation, the patient, if an adult, is given one-quarter of a grain of morphine and one-hundredth of a grain of atropine hypodermatically. The patient is placed on the table lying on the unaffected side, the arm of the affected side is placed above the head, thereby raising the scapula. The affected side is painted with iodine which is removed with alcohol. In adult cases a 1 per cent. solution of novocaine with adrenalin is used to infiltrate the line of incision. The incision is made over and parallel to the eighth rib in the posterior axillary line, and should be about four inches in length. About two inches of the eighth and ninth ribs are removed subperiosteally. In cases where there is a large flat rib only the ninth rib is removed. The pleura is opened by a stab with closed scissors. A piece of gauze is placed over the opening and after a short time the pleura is opened as widely as the incision will permit. The thin pus is then removed by a suction apparatus. The thick pus and the masses of fibrin, as is found in most instances, are removed by packing with plain gauze. This procedure may have to be repeated two or three times until the cavity appears to be free from fluid and fibrinous exudate. After this cleaning the pleural sac is carefully, thoroughly, and firmly packed with one and one-half inch washed iodoform gauze. In the average adult about fifteen yards of gauze is necessary for adequate packing. By adequate packing he meant the entire pleural cavity to be filled. The edges of the skin and muscle wounds are separated by packing tightly with plain gauze, thus fixing the muscles and skin as far apart as possible. If this is done there will be no excessive granulations in the wound and the subsequent dressings are much

less painful. He had learned from experience that the pain from these subsequent dressings is not from the pleural cavity but from passing the gauze over the raw surfaces between the skin and the cavity. The repacking can be made practically painless if there are two small retractors placed, one at the upper and one at the lower borders of the incision, about midway. In none of his cases had he found it necessary to use anæsthesia for the repacking, and in most instances after the second packing there is little or no discomfort experienced by the patient. Post-operatively, the patient is placed in bed in a semi-recumbent position and is encouraged to lie upon the affected side as much as possible. Morphine is used if indicated. At the end of three days the packing is removed and an X-ray is taken and the wound is repacked in the same manner but a lesser amount of gauze will be required at every subsequent dressing. The case is handled by changing the packing at intervals of two days until the wound is completely healed. A careful study of the expansion of the lung is made by X-ray. Pulmonary exercises by means of blow bottles are begun immediately after the first packing is removed, which encourages expansion. Fourteen of the cases thus treated were empyemas due to an infection with a pneumococcus, one was due to a streptococcus, one due to a pneumococcus in a patient with pulmonary tuberculosis. Eleven of these cases are entirely well. The first case was operated upon June 6, 1929, and has remained well ever since. He was seen in December, 1929, and has returned to his work. Four cases are still being treated and one case died two weeks after operation. This was the case of pulmonary tuberculosis. At the time of her death the operative wound was clean with a very small amount of discharge from the pleural cavity. No post-mortem was obtained. All of these patients were adults, except two. One was two years and three months old and, the other, this boy presented. In the two- and fourteen-year-old cases nitrous oxide and oxygen was the anæsthetic employed. In only one case was there any difficulty experienced when the pleura was opened. This case became dyspnœic, deeply cyanosed and the pulse rate rose from 110 to 140. He had a short jerky cough. The thoracotomy wound was covered with a piece of gauze for a few minutes when he suddenly expelled a large amount of mucus after which his respiratory distress cleared up. The amount of gauze used in these cases varied, in the adult the smallest amount being ten yards and the greatest amount twenty yards. The wounds were packed from six to twelve times before they were completely healed. In the two-year-old child a mistake was made for he was packed but three times and as his temperature remained flat he appeared well, had a good appetite, playing around the ward, when after eight days the temperature rose to 102, the pulse to 140. The X-rays showed that a small pocket of fluid was just above the closed sinus which necessitated a second operation. In another case, a young adult, the same condition arose. This will not happen again since his experience has increased. After the third or fourth packing the cavities were inspected by flashlight illumination and cystoscope and were all found to be clean and none of that thick, fibrous covering of the visceral pleura was in evidence. According to X-ray studies the subsequent expansion of the lung on the affected side progressed satisfactorily. No study of the tidal air changes was made. The condition of the wound at the time of discharge from the hospital showed that it was entirely healed in all but one case. This case had a small sinus and on X-ray a small pocket of fluid was found. The patient refused to have it removed but since that time he has returned and this small collection of fluid has disappeared. The period of stay in the hospital varied from eighteen to thirty-one days; the average stay being

about twenty-one days. At present there is a patient under treatment whose wound has been packed sixteen times but there is a complicating factor in this case of a gangrene of the pleura. On account of the odor it was deemed advisable to dress him almost daily. Doctor Connors believes that the advantages of this method of treatment are:

1. The opening in the chest wall is sufficiently large to allow complete evacuation of all pus and fibrinous exudate and later a thorough cleaning of the cavity.

2. Packing with iodoform gauze, which when properly done prevents the pocketing of pus, which has been the great trouble in the management of these cases. With the packing, adhesions are formed very early around the periphery of the packed cavity.

3. Secondary operations, if necessary, introduce no possible dangers of mediastinal flutter as there is an excellent fixation of the mediastinum.

4. It is in accord with sound surgical principles, namely, it affords adequate drainage of pus, producing sterilization of and obliteration of the cavity.

The question of mediastinal flutter may justly be raised, resulting from the production of an open pneumothorax when such a comparatively large opening is made into the pleural sac and death may occur from this cause alone. It is generally agreed that in empyema the mediastinum is fixed by adhesions, except those of streptococcic origin and the causes of death in the operative cases of streptococcic origin are laid at the door of mediastinal flutter which is not, in his opinion, the sole factor to be considered. In fact, death occurs in two groups of cases; in the first group death occurs at once while the patient is on the operating table or shortly after the operation and in this group of cases mediastinal flutter is a factor but collapse of the lung plays a large part in the dsypnœic condition of the patient by the forcing of the contents into the opposite bronchus, thereby producing collapse of the opposite lung as is shown by the return to better breathing after expelling the bronchial contents. His conclusions of this type of case are based upon a rather large experience with open traumatic wounds of the chest. He was satisfied that the phenomenon of a flexible mediastinum does not adequately explain the situation. In the second group of cases in which death occurs in from four to thirty days after operation death is due to an improper evacuation and drainage of pus which produces a pyemia.

Dr. John Douglas said that if Doctor Connors had a method of treatment of empyema by which he can get the patients well and able to leave the hospital in less than twenty-one days, it is better than any other so far as he knows. The object of treatment in empyema is the removal of infection, and drainage and sterilization of the cavity should be effected as soon as possible before the visceral pleura becomes thickened and prevents lung expansion. Doctor Connors' method certainly provides adequate drainage, and it would appear that he has found a way of lessening air pressure from outside so that the lung can continue to expand. Doctor Douglas also agreed that local anæsthesia should be used, instead of general, in all adult cases of empyema. Doctor Douglas believed that the shorter time in which private patients get well, than do the ward cases of empyema, is adequate proof that the after-care is almost as important as the primary operation; perhaps the shorter time in Doctor Connors' cases is due to his having taken care of them himself because of his especial interest in this series. Regarding the choice

between the two methods, the open and the closed, Doctor Douglas believed the closed method with Dakin's irrigation, using the two-way tube, offers advantages over the open method, and he also believed this method of Doctor Connors' had an advantage over the old open method. The speaker asked Doctor Connors two questions: one, whether there had been any signs of iodoform poisoning or any bad effects from using so many yards of iodoform gauze in the packing of these cases. Of course in some cases this is due to an idiosyncrasy, but there have been severe cases of poisoning which resulted from simply using iodoform on an infected finger, a marked rash and swelling extending up the arm, and even cerebral symptoms have developed after the use of a very small quantity. Doctor Douglas used iodoform gauze with caution; even in resection for carcinoma of the rectum where there was a large cavity in the pelvis, he supplemented the use of it with plain gauze. The second question was: in the case of lung abscess did Doctor Connors penetrate to the abscess through normal pleura in a one-stage operation, without any attempt to block off the pleura?

Doctor Connors, in reply, said that in the entire series of cases there had been no symptoms of iodoform poisoning; this was probably due to the fact that the gauze was always washed before insertion into the cavity. He agreed thoroughly with Doctor Douglas as to the value of the after-care as a factor in securing the earlier cure of these cases. There were no signs of mediastinal flutter in any of the empyema cases. They were carefully watched and the only change observed was a gasp-like inspiration at the moment the chest was opened. There was a rise in the pulse rate and a cough which lasted only a minute, after which the patient seemed to experience no untoward effects. In the case of abscess of the upper lobe of the lung there was some flutter which interfered with the pulse and respiration, so much so that it was necessary to resort to the use of oxygen, but after forty-eight hours recovery was uneventful. The later cases have been done with the patient placed in almost a sitting position and with these cases there has been less difficulty than with those lying flat on the unaffected side.

## THE CAUSES OF DEATH AFTER OPERATION. A STUDY BASED ON EIGHT HUNDRED AUTOPSIES

Dr. Harold Neuhof read a paper with the above title for which see page 321.

Dr. Howard Lilienthal said that the number of autopsies in this series, eight hundred, was impressive, and he felt that any mistakes that might have been made in Doctor Neuhof's analysis was counterbalanced by the size of the collection of cases which has been studied so carefully. Excluding the forty-eight hour ones was generous for the speaker knew that some of them had died of suppuration, or of an infection which would have gone on to suppuration. (By the by, it was noticeable that Doctor Neuhof had avoided using the word "infection.") A patient may develop violent erysipelas and die in forty-eight hours; all surgeons had seen this happen. In

Table IV, the Genito-Urinary Table, it was astonishing to see no record of deaths from pneumonia in the suppurative cases; this was the more remarkable because deaths from pneumonia in non-suppurative cases were recorded. Possibly the explanation is that the suppurative cases did not live long enough. Regarding Table VI, Exploratory Laparotomy, Doctor Lilienthal was not certain what Doctor Neuhof meant by the term "exploratory laparotomy," in this study. The speaker had always regarded the procedure as uncertain and dubious; Dr. Morris Manges had once said that exploratory laparotomy was among the most dangerous operations of surgery. If one opens the abdomen and nothing is found, the operation is rarely severe and it is doubtful if such patients would die of pneumonia or sepsis in large numbers. If a so-called exploratory operation is done and it is found that there is a pathological condition to be relieved, it is no longer an "exploratory" procedure. It was in regard to the bad cases where exploratory operation had to be done that Doctor Lilienthal was in doubt; Doctor Neuhof had listed 21 per cent. of what the speaker regarded as suppurative cases in which death had followed "exploratory" laparotomy.

Doctor Neuhof's general classification seemed very fair; there are some cases in which death can occur from infection but other causes are also present. Doctor Neuhof had not considered diseases of the chest, such as tuberculosis, where other dangers besides suppuration threatened. Thoracic surgery should be included in a study of this kind for more and more work was being done in this field.

As to the lesson to be learned from this essay, the most important was that lives can be saved if during the post-operative period one looks for the most natural thing that might cause death, and that has been shown by Doctor Neuhof to be suppuration. Consequently, when things are not going well in cases that ought to be doing well, look at the lungs and look for suppuration. When an automobile fails on the road one looks in the gas tank first.

Dr. Richard Lewisohn said that there seemed to be a doubt among surgeons as to what one could really call a clean case and what one could call an unclean case. A hernia, a goitre or a brain tumor must be regarded as a clean case. The speaker would not regard as a primary clean case an operation on the bladder, or on the gastro-intestinal tract where an opening must be made into a gut containing infective material and where the surgeon lays the patient open to infection, because there can be no adequate protection. Doctor Lewisohn did not think one could regard such a case as a clean one from the standpoint of possible surgical infection. Everyone knows that in former years the importance of peritoneal infection was under-valued and in case of death the chest was accused. A leading surgeon once said that if there was no post-mortem on a case of death following an abdominal operation it would always be ascribed to pneumonia. Doctor Lewisohn believed that it was very important that Doctor Neuhof had found peritoneal infection to be so often the cause of death after laparotomy. The speaker had occasion many years ago, when he was an assistant in Doctor Czerny's

clinic, to look up the causes of death in patients suffering from post-operative pneumonia from 1901 to 1905. He found that in 50 per cent. of the deaths where post-operative pneumonia was found at the post-mortem examination, the post-operative pneumonia had been caused by an infection in the abdomen spreading up through the diaphragm. Doctor Neuhof has gone a step farther and has shown that in many cases one is not dealing with pneumonia at all, but with a suppuration in the operative field. As to Doctor Lilienthal's hope that if one can make the diagnosis of peritonitis early enough, re-operation can cure the patient, Doctor Lewisohn doubted if that were so. Secondary operations can cure the patients in the presence of a well-localized abscess. They fail to effect a cure in the presence of a spreading peritonitis without localization.

Doctor Hyman expressed surprise to see in Table IV, the Urological Section, that in a series of fifty-five deaths, 44 per cent. were due to post-operative suppuration in the upper urinary tract. He believed that one would be apt to get an erroneous opinion from this. Doctor Neuhof, in reading the paper, had changed the nomenclatures "infective" and "non-infective" to "suppurative" and "non-suppurative." One could get the idea from the paper that 44 per cent. of the non-infective urological cases died of suppuration in one form or another. Of course, it comes down to the question just where to stop in saying a patient is infected or not. Practically all the prostate cases are infected. The speaker had an opportunity to see the work sheets of this table. Prostatic adenoma, carcinoma of the bladder or vesical calculus, and so on; at least 95 per cent. of them are infected cases. One rarely sees clear urine in prostatics coming for operation. In talking this over with Doctor Neuhof he said that he wanted to get away from the words "infected" and "non-infected," but confusion as to these terms is likely to lead to error in considering the upper urinary tract. The kidneys and ureters are generally not catheterized prior to operation in this type of case and one cannot therefore tell the degree of infection present before operation. Two of these cases of prostate adenoma showed at autopsy an infected kidney and hydroureter. This condition was surely present at the time of operation. Doctor Hyman said that what he wanted to bring out was that, according to this classification, an erroneous idea as to the frequency of suppuration as the cause of death in non-suppurative urological conditions may be obtained. The cases do die of suppuration, but in 80 or 90 per cent. a primary suppurative condition has existed in the urinary tract before operation.

Dr. John Douglas referred to one point in Doctor Neuhof's tables that had not yet been discussed: in 204 deaths in non-suppurative cases, 39 per cent. of the deaths occurred from suppuration. Also, in 50 per cent. of the cases which died of suppuration, suppuration was not even suspected as the cause of death, according to another table. Doctor Neuhof had also called attention to the fact that although the ante-mortem diagnosis of pneumonia was made in a considerable number of cases, in only 60 per cent. was the

diagnosis of pneumonia confirmed, so that in 40 per cent. it was wrong. The diagnosis of intra-abdominal suppuration depends on the symptoms of local tenderness, localized pain and rigidity. Some patients are very sick, have no rigidity, the pain is not local, and they are tender all over or too sick to be tender enough for diagnosis; they often have a chest condition, due to cardiac failure or a hyperstatic congestion which it is difficult to differentiate from pneumonia. In such cases, how can one make a diagnosis of suppuration? The question is, what of greatest importance can be learned for the benefit of such cases from this presentation of Doctor Neuhof's? In the speaker's opinion the presumptive evidence would be, in these cases that are going badly, that they have infection and suppuration; if it is localized, there may be a chance to do something, and if it is general, they will die anyway.

Dr. Leo Mayer (by invitation) presented a report which he had prepared showing the cause of death in ten orthopædic cases—all bone and joint cases. Of the series there was only one in which there was frank suppuration. One death was from tuberculous meningitis following a fusion operation on the spine; four died of acute septicæmia following acute osteomyelitis; one died of pulmonary embolism; three died following malignant growths; and one died of acute suppuration following arthroplasty of the knee. It would, therefore, seem that the figures in orthopædic surgery were not in accord with those of surgery of other parts of the body.

Doctor Neuhof, in closing the discussion, answered Doctor Lilienthal's question as to what was meant by exploratory laparotomy by stating that this comprises a group of cases in which at operation the patients were found to be suffering from an irremovable neoplasm and upon whom nothing further was done than exploration. There were some chest cases in this series of 800 in which thoracoplasty was done for pulmonary tuberculosis, but these patients have been classified as having died of their original disease, *viz.*, tuberculosis. As to the nomenclatures "infected" and "non-infected" *versus* "suppurative" and "non-suppurative," this is not of great importance; Doctor Hyman was correct in stating that the speaker had previously used the first mentioned terms. As a matter of fact he had changed the nomenclature a number of times, but had finally come to the conclusion that "suppurative" and "non-suppurative" would be more inclusive as these terms would embrace some of the doubtful cases. As Doctor Lewisohn said, it is important to know when a case is "clean," and this is a subject that has been discussed pro and con at many surgical conferences without having been finally settled. In this series, if a patient were an interval case and had died after the operation, say from appendicitis in which the appendix had been found to be not acutely inflamed, the case was not placed in the suppurative column. In a similar way the urinary cases have been classified; most of them were prostate cases. When death was ascribed to suppuration the evidence had to be clear that the suppuration was recent if the case were to be classed as death from suppuration.

# BRIEF COMMUNICATIONS

## THE VALUE OF SPALTEHOLZ CLEARING METHOD IN THE STUDY OF SURGICAL AND PATHOLOGICAL SPECIMENS

THIS communication has been prompted by many inquiries as to the technic of preparing large cleared sections of tissue for study in our laboratory of surgical pathology.

The Spalteholz method which renders tissues transparent has long been of inestimable value in experimental work. Its application in the field of pathology and as an adjunct to teaching was first suggested by a study of the beautiful and instructive histological sections of whole breasts prepared by Sir Lenthal Cheatle.[1] These were given to us for teaching purposes some years ago and lacking the equipment for their preparation we resorted to the Spalteholz clearing method.

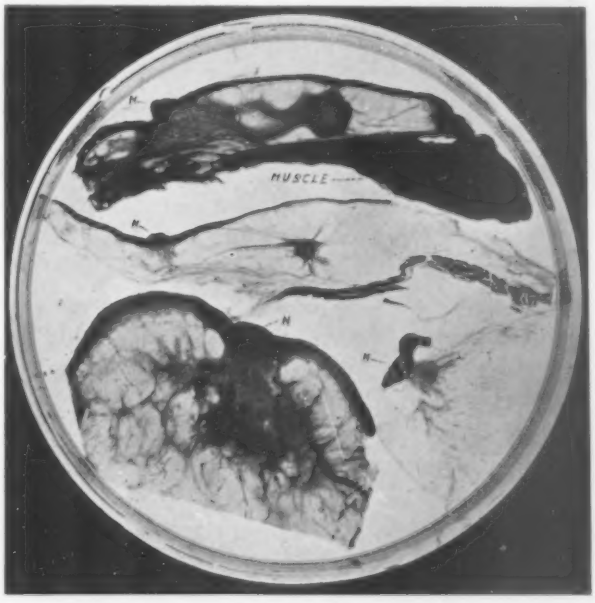


FIG. 1.—Photograph by trans-illumination of four cleared specimens of mammary carcinoma. The upper specimen is two centimetres in thickness. N indicates nipple.

The original Spalteholz technic of fixation by formalin has been described by Sabin[2] with an added modification of Carnoy's fluid used as a fixative in her work on mammalian embryos. The essentials of the Spalteholz method are, "first, fixation in formalin; second, a thorough bleaching of the tissues with hydrogen peroxide to remove the hæmoglobin and other pigments; third, dehydration; and fourth, clearing the specimens in an oil which has the same index of refraction as the tissues." Ordinarily a considerable amount of blood is removed in the handling of fresh specimens and what little remains after clearing serves to identify the blood vessels at a glance. Complete removal of the hæmoglobin may be accomplished by bleaching the fixed specimen in undiluted hydrogen peroxide, the time required being from fifteen to thirty minutes. "Following the bleaching the specimen must be washed thoroughly in running water and in distilled water. The dehydration may be begun with 50 per cent. alcohol and the percentage increased successively by five points or less. After two changes of a good grade of absolute alcohol, the specimens are passed through two changes of benzene into the synthetic

oil of wintergreen. The small amount of benzene which is carried over evaporates quickly, and the few bubbles which develop in the bleaching process can be removed with needles. The oil of wintergreen should be entirely colorless but both the specimens and the oil will gradually become brown with age," and should be protected from sunlight. "They can be returned to alcohol for storage and recleared when desired or they may be made permanent in balsam." Keeping them in the oil of wintergreen so toughens the tissues that they can be dissected under the low- or high-power binocular microscope.

Microscopic study of these cleared specimens is best made by submersion in oil in a flat glass dish beneath which is a substage lamp or frosted electric light globe, thus transilluminating the tissue. For histological study any portion of the specimen can be removed with a safety razor blade, passed through a mixture of oil of wintergreen and paraffin, and then embedded in paraffin for section.

Routine pathological studies are of course always made on other portions of the fresh specimen.

Sabin's use of Carnoy's mixture for fixation has further simplified this method. The mixture "is absolute alcohol 60 parts, chloroform 30 parts, and glacial acetic acid 10 parts. In this mixture the acid is sufficiently strong to bleach the hæmoglobin so that the peroxide is unnecessary. The penetrating power of the fixative is very great. . . . The relations of the tissues are well maintained and the swelling due to the acetic acid tends to counteract the shrinkage that always takes place in the oil of wintergreen. The fixative does not affect any of the injection fluids. The process after fixation in the Carnoy's mixture is simple; the specimens remain in the fixative from 2 to 12 hours and are then placed directly into 70 per cent. alcohol, dehydrated in graded alcohols, and cleared as before."

With very large specimens it is sometimes better to take the fresh material obtained from the operating room or at autopsy, freeze it in its natural shape by placing it in a refrigerator plant and then cut sections of from 0.5 to 20 centimetres in thickness, which on thawing are placed in a fixing solution and then cleared.

The accompanying photograph (Fig. 1) of four specimens of mammary carcinoma was made by transillumination. The transparency, the thickness of the specimens (0.5 to 2.0 centimetres) and the finer details are poorly shown but can readily be seen in their three dimensions with the naked eye or with a binocular lens. These four specimens were obtained by cutting through the tissue removed in a radical amputation of the breast so as to pass through both the tumor and the nipple (N). Two of the specimens show the malignancy beneath the nipple, the others show the carcinoma as more circumscribed and of varying characteristics at some distance from the nipple. The fatty tissue has become so transparent that it sharply outlines the fibrous and cancerous tissues and clearly reveals the spread of the malignant cells.

These whole breast sections afford a visual picture of great value in the

study of tumors, and of their influence on the skin, the breast tissue and the underlying muscle. Frequently three or four such sections, 2 centimeteres or more in thickness, suffice to present the entire tumor process surrounded by transparent breast tissue. The method has been extended to material other than from the breast and is useful in the study of specimens from the brain, lungs, intestinal tract and integument. These specimens serve as a permanent mount in the museum and have also been of great value in our teaching.

### CONCLUSION

The Spalteholz clearing method and Sabin's modification require no special apparatus and offer a simple and satisfactory technic in the preparation of surgical and pathological specimens.

By their transparency these specimens provide for further study of tumors and disease areas in relation to the surrounding structures.

In conjunction with histological preparations several such sections suffice to convey a complete picture of a given pathological process and are of inestimable value in teaching.

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## DOUBLE GALL-BLADDER

THIRTEEN years ago Dr. August Schachner[1] of Louisville, Kentucky, reported a case of a double gall-bladder (ANNALS OF SURGERY, October, 1916).

I desire now to place on record another case similar to the one presented in his paper. In an exhaustive study of the literature I have been able to find to date only seven cases of double gall-bladder reported. The specimen now described was shown at last year's meeting of the American Gastro-Enterological Association, at Washington, D. C., and brought forth considerable comment because of its rarity.

In a consideration of congenital anomalies of the liver and gall-bladder it has been stated that two livers in a human being is the rarest condition, there being only one such case on record. The next rarest anomaly is reported as being absence of the gall-bladder and all ducts, with thirteen cases being reported. This being true, then absence of the gall-bladder and all ducts is not the second rarest anomaly but rather double gall-bladder is the second rarest, inasmuch as only seven cases have been reported, this

case being the eighth. This would place the third rarest anomaly as absence of the gall-bladder and all ducts. The next, or fourth rarest anomaly is absence of gall-bladder, cystic and common ducts, with fourteen cases being reported, and next, or fifth rarest is absence of gall-bladder and cystic duct, there being thirty-one cases reported. True left-sided gall-bladder has been reported many times.

Double gall-bladder in animals has not been very often reported. Double gall-bladders with separate cystic ducts has been reported in two young rams.[2] In each case the gall-bladder was as large as usual and the chambers were separated by septa of connective tissue. In one case, the both chambers were equal in size, while in the other case, the one chamber was only half the size of the other. All chambers had bile and there were no liver anomalies. A similar condition was also reported in a five-year-old ox where one chamber was only one-fourth the size of the other and where the septum was perforated, allowing an intercommunication.

Doctor Schachner's case[3] in the human had two unequal chambers with separate cystic ducts and stones in each side and in this respect was similar to the case here reported.

Dr. Benjamin W. Rycroft, of Beacon House, Bradford, England, very kindly forwarded me a diagram illustrating his case[4] of Bilocular gall-bladder. Examination of this sketch reveals the fact that his case was not one of complete double gall-bladder inasmuch as the septum extended from the fundus only three-fourths way down to the neck of the gall-bladder. There were twelve calculi present in this gall-bladder.

Professor C. F. Van Oyen, of Utrecht, forwarded me a description of the case reported by Dr. C. DeGraaf[5] in which the patient had two distinctly separate gall-bladders and two separate cystic ducts, each one centimetre long, which united to form one cystic duct.

H. Critchley Hinder, M.B., Ch.M., Hon. Surgeon, Royal Prince Alfred Hospital, Sydney, Australia, reported a case[6] wherein the septum extended half way up the fundus from the neck exactly opposite to the position of the septum in the case reported by Doctor Rycroft. This latter case also had stones in both loculi. Those interested in the anomaly would do well to consult his paper.

Case Report.—Mrs. B. F., aged thirty years, was referred to me at the Locust Mountain Hospital, April 15, 1928, by Dr. Ivor D. Fenton, of Mahanoy City, because of severe pain in the upper right quadrant of the abdomen. Her temperature, pulse and respiration on admission was 99–98–20. The first attack was seven years ago accompanied by a chill and weakness and she had many attacks in this interval. Her pains were not intermittent and always lasted until a hypodermic of morphia was given. These attacks were usually at night and the interval of time between attacks became less toward the end. Patient states she was extremely hungry for all foods all the time but could not eat when food was placed before her.

The last attack of pain was intermittent, started three days ago, and the patient had chills with nausea and vomiting each of these days. The early history of the patient added nothing to the diagnosis, although it might be here stated that on examination, a positive blood serology was found.

The head, mouth, and throat were normal; there was no jaundice, and the lungs were clear. The heart sounds were of good quality and murmurs were not heard. The abdomen was tense and there was rigidity over the upper right rectus. Murphy's sign was positive; no masses were palpable, and Carnett's both A and B tests for neuralgia of the abdominal wall were negative. She was admitted with a tentative diagnosis of cholelithiasis.

Iodeikon was administered intravenously that evening for visualization of the gall-bladder and the X-ray report by Dr. P. B. Mulligan was as follows:

"No gall-bladder shadow was evident on any of the films of this series. There is, however, a shadow opposite the second and third lumbar vertebræ which is about the size of an English walnut and which has much the appearance of being one large gall-stone."

I operated upon the patient April 26th. When I attempted to isolate the cystic ducts, I found two of them, each of which I ligated separately. I was impressed with

FIG. 1.—Double gall-bladder showing serosal surface and depression or ridge of the septa and beginning of each separate cystic duct.

FIG. 2.—Double gall-bladder specimen divided sagitally, showing strawberry mucosal surface. Note exit of each cystic duct and septum.

a hard white ridge extending upward on the gall-bladder and in view of the fact that I had two cystic ducts, became suspicious that there was a septum present in the gall-bladder. This proved to be true. Each side was tensely full of bile of the B type and there were numerous small stones present in each chamber and one very large stone in one of the chambers.

The gall-bladder was removed *in toto* and a soft slit rubber tube and wick inserted. She made a very rapid convalescence and was discharged from the hospital May 15, 1928. Follow-up reports from her family physician state that she is enjoying very good health and has had no further trouble.

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# BOOK REVIEW

STONE AND CALCULOUS DISEASE OF THE URINARY ORGANS, by J. SWIFT JOLY, M.D. 8 vo.; cloth; pp. 555; illustrations, 193. C. V. Mosby Co., St. Louis, Mo., 1929.

Extraordinary as it may seem, there has been no book published during the past twenty years which has been devoted exclusively to the question of urinary calculi. Certainly the chapters devoted to its consideration which appear in books on general urology and more particularly in general surgery must necessarily be abbreviated and but in a small measure represent the numerous and varied phases of the subject in its entirety.

In the present volume, the subject matter is presented in a peculiarly readable form. The introductory chapter on the history of urinary lithiasis is of the greatest value to any practitioner and most interestingly narrated. The succeeding chapters on the general characteristics of calculi and their etiology are complete in their detail and will repay the perusal of any physician in general practice as well as of one devoting himself to this specialty.

In the consideration of renal and ureteral calculi, the author continually emphasizes the necessity of conservative surgery and considers each debated question in such detail that its merits are readily appreciable. A most instructive consideration of the medical treatment, prevention, non-operative and post-operative, is included. The topic of differential diagnosis is excellently handled. It is interesting to note the omission of the occurrence of torsion of the kidney (Dietl's crises) in this connection.

Plastic operations on dilated pelves, a procedure which has received impetus lately, is not taken up and might well be included. The author's preference for the lower median incision for exposure of the pelvic ureter in extraperitoneal ureterotomy rather than the lateral, is noted and its consideration is well worth while studying.

The alarming complication of calculous anuria and the indications for operative or non-operative interference is a most important inclusion in the present work and analysis of it will repay both the urologist and the general surgeon.

Certainly the revolutionary methods of investigation of the genito-urinary tract for disease chiefly by the use of radiography, cystoscopy and the application of the recent advances in physiological chemistry have necessitated a complete revision of our knowledge of procedure in these conditions and the author has succeeded admirably in presenting the subject in its entirety in a most attractive way.

JAMES T. PILCHER, M.D.

# MEMOIR

## FRANCIS JOHN SHEPHERD, M.D., C.M., L.L.D., F.R.C.S.

1851—1929

FRANCIS JOHN SHEPHERD was born at Vaudreuil, near Montreal. His early education was received at the Montreal High School, followed by a year in the Arts Faculty of McGill University before entering the Medical Faculty from which he graduated in 1873. He then went abroad for further study in London, Paris, and Vienna. In the latter city he was joined by William Osler who had graduated from McGill the year before.

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Two outstanding features of this trip were his concentration on anatomy and the methods employed in teaching anatomy in England and on the Continent, and his interest in dermatology. He took a course under Hebra and gave special attention to dermatology throughout his professional career.

On his return to Montreal he was appointed Demonstrator of Anatomy in the Medical Faculty of McGill University and, on the death of Professor Scott, was made full professor and occupied the chair for thirty years. Under his direction the teaching of human and comparative anatomy attained a very high standard. He gradually assembled in the Anatomical Museum a large and rare collection of anatomical anomalies which, unfortunately, was lost in the fire that destroyed the building in which they were housed. Many of the most brilliant and successful teachers in medicine and surgery and the specialties in McGill received their early training and inspiration from Doctor Shepherd.

As Demonstrator of Anatomy, Doctor Shepherd organized a clinic in dermatology in the Montreal General Hospital in which he took great interest.

As a surgeon Doctor Shepherd was a most careful and conscientious diagnostician and operator. He established a Summer Session at the Montreal General Hospital which he carried on for many years.

His contributions to surgical literature were numerous. He was for many years on the Editorial Board of the Montreal Medical Journal, and contributed a monthly review of surgical publications, terse, critical and sometimes humorous. These were afterwards published in book form. He was the author of the article on the "Thyroid" in the *American Practice of Surgery;* joint author of *American Text-book of Surgery;* and contributed articles on Anatomy and Dermatology for Wood's *Reference Handbook of the Medical Sciences.* He was President of the Montreal Medico-Chirurgical Society in 1900 and President of the Canadian Medical Association in 1901.

Doctor Shepherd's character and scientific work were very widely recognized. He was an Honorary Fellow of the Royal College of Surgeons, Edinburgh, and a Doctor of Laws, Edinburgh University; L.L.D. Harvard, 1906; L.L.D. McGill, 1915; and L.L.D. Queen's, 1919. He received the Honorary Fellowship of the Royal College of Surgeons in 1913 and Honorary Fellowship of the American College of Surgeons in 1914. He was corresponding member of the Société Internationale de Chirurgie de Paris, and foreign member of the American Academy of Arts and Sciences. On his attaining his fiftieth year after graduation, he was tendered a banquet by his former house surgeons and demonstrators of anatomy, and presented with a fine piece of Georgian silver. The following year his numerous friends in the profession presented him with a portrait of himself by Miss Des Clayes.

Doctor Shepherd was fond of literature and art and was a recognized critic of oil paintings. He was twice president of the Montreal Art Association. For many years he was a member of the Board of Trustees of the National Gallery at Ottawa and Chairman of the Board at the time of his death.

Doctor Shepherd had two daughters, and one son who was a war casualty.

Among McGill's distinguished graduates, Doctor Shepherd's name stands high. His influence in the profession and socially was very great. He always stood for what was pure and noble, and he set his face definitely against all commercializing tendencies and always had the full confidence of his confreres and especially of the younger men who looked up to him for guidance.

GEORGE E. ARMSTRONG

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